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VOL. II.—31ST YEAR.

SYDNEY, SATURDAY, DECEMBER 9, 1944.

No. 24.

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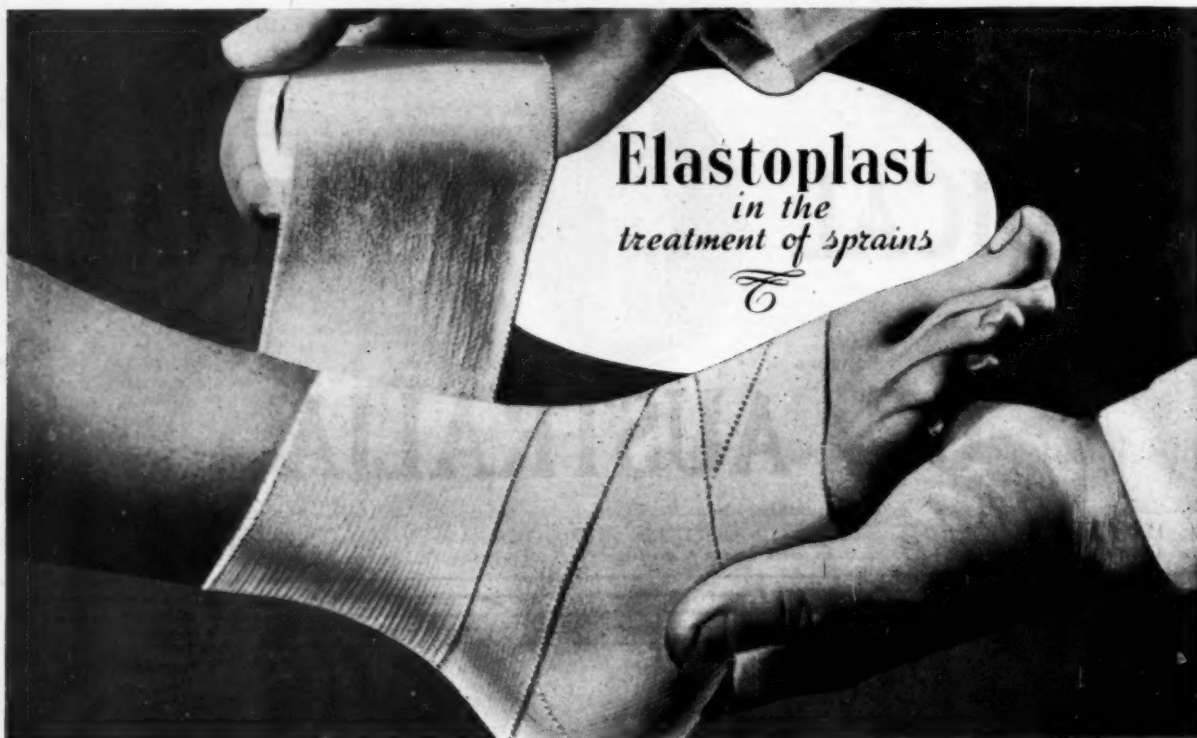
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G. E. Rennie Memorial Lecture.¹

TORULA INFECTION OF THE CENTRAL NERVOUS SYSTEM.

By C. T. CHAMPION DE CRESPIGNY,
Adelaide.

AN invitation to give this lecture before our College is an honour too great for any conventional words of acknowledgement; but I may be permitted to offer my thanks to my colleagues, and to say that, even if it had been possible to decline a request which came with more than the force of a command, both inclination and duty would have impelled me to take the opportunity to honour the name and memory of a great Australian physician and teacher of medicine.

George Edward Rennie, whom this lecture commemorates, was born on September 10, 1861, and died on August 10, 1923.

He had a brilliant scholastic career, crowned by the award of the gold medal in his examination for the M.D. of London. He became a member of the Royal College of Physicians in 1899 and was elected Fellow eight years later. He was both scholar and scientist, for he had taken his degree in arts with first-class honours in Greek and Latin.

Although he practised medicine in all its branches, his main study was neurology. For this he was particularly well equipped. Neurology is a science which requires on the part of its student a thorough knowledge of anatomy—gross and microscopic—physiology and pathology. Rennie was a man of deep learning and wide experience in these

basic sciences, and it is said that he astonished his students when confronted with a rare nervous disease by his intimate acquaintance with its structural and functional basis. His mind was of the logical and inquiring nature which has been the gift of Hughlings Jackson and other great masters of English neurology.

I had the pleasure of meeting Dr. Rennie on only one occasion, so that I can claim no great knowledge of the man himself except through his reputation.

His presence was austere and his intense concentration on his art was apparent. This I remember well, because when he visited Adelaide in 1920, I think, I showed him a case which was interesting me at the time. It was during the *encephalitis lethargica* epidemic. The patient, a semi-comatose young man, exhibited constant involuntary and incoordinate movements of the trunk and limbs. We agreed that we had not seen the condition before. A few weeks later in *The Lancet* appeared the description of a case of the myoclonic form of *encephalitis lethargica*.

It is said that Rennie seldom smiled. However, Professor Cleland has told me that he himself evoked a smile from Dr. Rennie by the macabre remark that the best hospitals had the greatest number of deaths. It must be remembered that Professor Cleland takes the morbid anatomists' point of view.

Dr. Rennie devoted his whole life to his work. His mind was cast in a mould which made him hate all pettiness and unworthy actions. His brother, the late Edward Henry Rennie, who occupied the Chair of Chemistry in the University of Adelaide and was one of the founders of its medical school, resembled him in the power of his intellect and the simplicity and austerity of his life. He is always remembered with respect and affection by his students.

The lives of such men are valuable by their example beyond their written or even spoken words.

I have chosen my subject because Dr. Rennie was a general physician with a special interest in neurology, and therefore I propose to speak of a condition which concerns both the neurologist and the general physician.

¹Delivered before the Royal Australasian College of Physicians at Sydney on April 21, 1944.

NOMENCLATURE IN TORULA INFECTION OF THE CENTRAL NERVOUS SYSTEM.

The nomenclature of the yeasts pathogenic for man has caused confusion in the literature and is still debated. To Dodge's "Medical Mycology" I am indebted for the following account.

Of those capable of causing systemic infection, by far the most important belong to the genus *Cryptococcus*. The was named *cryptococcus* by Kuetzer in 1833. The name *torula* was bestowed by Turpin in 1796, but his *torulae* probably included sporogenous as well as asporogenous strains.

The characteristics of the genus *Cryptococcus* are as follows: the cells are spherical, ovoid or ellipsoid, occurring singly or held in more or less irregular groups by the secretion of thick gelified capsules, especially in old age. These do not form ascospores, mycelium or pseudomycelium. On liquid media a thick pellicle is formed by the coalescence of slimy floating islets. When present, the sediment is usually slimy. There is no fermentation, and acid formation is rare with carbohydrates. Liquefaction with gelatine is very slow or absent.

Two groups of organisms stand out as the principal pathogens of this genus: (i) The first group was isolated from tumours and intensively studied while the yeast theory of the origin of cancer was in the ascendant and utterly ignored since this theory was discarded. (ii) The other important group centres about *Cryptococcus histolytica*, which has been proved repeatedly to be the morbid agent in *torula meningitis*.⁽¹⁾

Because the name *torula* has been used in the literature much more frequently than *cryptococcus*, I have retained it in the title of this address. Blastomycosis has been used also by several writers to designate general infection by yeasts or blastomyces including *torulae*. Since it does not cause fermentation, *cryptococcus* or *torula* belongs to the group of pseudo-yeasts.

DISTRIBUTION.

Torulosis is a rare disease. Up to the present rather less than 100 cases seem to have been described in medical literature.

Yeasts occur in nearly all parts of the world, and it seems that the pathogenic members of the group are equally widely distributed. Cases have been recorded in Europe, North and South America, Palestine, South Africa and Java. Australia has had a larger share than its population warrants, for up to the present about ten cases have been recorded in the Commonwealth.

HISTORICAL.

Although the disease is so uncommon, it has produced a very large literature, and I shall indicate only a few salient landmarks in its history. Some of the cases quoted are not examples of true *torula* infection, but are due to other yeasts.

The first authentic case of yeast invasion of the brain seems to have been recorded in 1861 by Zenker in Dresden.⁽²⁾

A man was brought into hospital with symptoms of right-sided hemiplegia and died after two days. At post-mortem examination an extensive left-sided encephalitis was found with a loose spongy texture of the substance of the brain consisting of yellow softening and degeneration of the nervous tissue *et cetera*. Macroscopically on the cut surface of the brain there were numerous small sharply defined beads of pus which could be lifted out in their entirety with a needle. On microscopic examination they were found to consist of conglomerations of strings of fungi surrounded by a thin layer of pus. The fact that all beads of pus were showing the same structure indicated that the fungi were responsible for the pus by irritating the surrounding tissue. The fungi were also regarded as the cause of the encephalitis and not as a lesion secondary to existing encephalitis. It is of importance to state that on tongue and pharynx masses of *Oidium albicans* were found.

It is very suggestive that from here, after erosion of the surface, spores of the fungi came into the blood and the brain. A secondary invasion of the brain by fungi *post*

mortem was definitely excluded by carefully examining the fresh brain immediately after it was removed from the body.

No further cases of yeast infection seem to have been recognized until 1895, when O. Busse⁽³⁾ described a case under the title "About a Case of *Saccharomycosis Hominis*". This seems to be the first recorded case of systemic invasion by yeast, but the organism does not appear to have been a *cryptococcus*.

A woman, aged thirty-one years, developed a swelling of the tibia with a pre-clinical history of six months and died after five months of medical attention. The main clinical features were painful swelling and ulceration of the skin of the middle of the tibia, of the ulna, of the sixth rib, and of both knees; hæmaturia was also present. Only rarely did the temperature rise to 38.5° C. Post-mortem examination revealed generalized yeast infection with multiple abscesses and nodules in the tibia, ulna, skull, sixth rib, both kidneys, spleen, and both lungs with cavities in the lungs and pleurisy. The main histological features included abscess formation, central necrosis, and numerous giant cells with phagocytosis of yeast bodies. These were also free in the tissue. The yeast was found to ferment glucose and to stain with carbol fuchsin. It grew at from 10° to 38° C. In young cultures contours were single, in older cultures they were double (see Figure I). The yeast grew on gelatine, glycerine-agar, agar, blood serum, bouillon, potato, and decoction of plums; the colonies were milky white. The life of the colonies was three to five months. The size of the yeast bodies was 7-15µ. In dogs and rabbits the yeast caused local inflammation, but healing occurred within three weeks. In mice death took place in four to ten days with general infection after intramuscular inoculation.

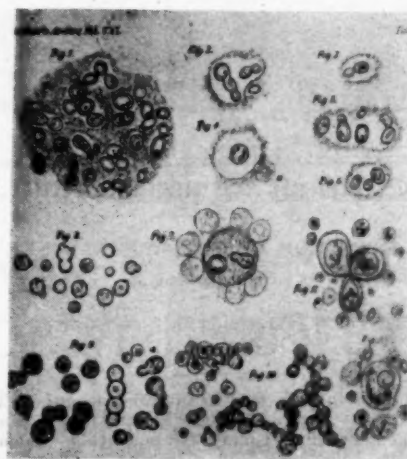


FIGURE I.

Drawings of *torula* cells. (Virchow's Archiv, 1895; Busse's case.)

In 1896 Gilchrist and Stokes described a case of chronic infection of the skin of eleven years' duration in a man, aged thirty-three.⁽⁴⁾

From the lesions were obtained numerous doubly contoured very refractile bodies. They showed budding, long branching mycelia with sporules, hyphae and knot-like projections. Dr. Flexner injected into the vein of a dog a pure culture teased in saline solution. Two months afterwards the animal was killed. Mucinous lesions were found in the lungs and pleurae. The bronchial glands were enlarged and bright yellow. The lesions contained yeast bodies. This yeast has been called *Torula Gilchristi*, and the name has been applied to other cases of torulosis. This seems an error, because the organism formed mycelium and hyphae in cultures. *Torula (Cryptococcus) histolytica* does not.

In 1905 von Hansemann in Berlin found yeast-like bodies in the cerebro-spinal fluid, but did not then recognize

them.⁽³⁾ Cultures could not be grown and no animal inoculations were made, but the brain showed "white antrium".

In 1907 the disease was first diagnosed during the life of the patient by Türk.⁽⁴⁾

A man with advanced pulmonary tuberculosis developed meningitic symptoms and was naturally believed to have tuberculous meningitis. When the cerebro-spinal fluid was submitted to the pathologist, Kretz, he at first thought that diabetic urine infected with yeast had been sent to him as a joke.

In a case reported by Versé in 1914, in addition to strictly meningeal involvement, there were foci in the liver, kidneys and spleen.⁽⁵⁾ This is the first report of torular meningitis with dissemination to other organs.

In 1930 Smith and Crawford reported the first case in the United Kingdom.⁽⁶⁾

In 1931 W. Freeman made a very full review of all the cases recorded until that date⁽⁷⁾ and added eight or nine more.

In 1942 Marshall and Teed reported a case of *Torula histolytica* meningo-encephalitis⁽⁸⁾ in which recovery followed bilateral mastoidectomy and sulphonamide (sulphadiazine, sulphonilamide derivative) therapy.

The patient was a girl, aged nine years. She developed meningitic symptoms. Double mastoidectomy was performed. Choke-disk appeared. Lumbar puncture showed yeast meningitis (*Torula histolytica* being found in the cerebro-spinal fluid). Sulphadiazine was administered by the mouth; 0.5 grammes every four hours kept the blood level at six milligrammes per 100 cubic centimetres, and in the cerebro-spinal fluid 4.75 milligrammes. The drug was given from January 15 to February 2. However, with its withdrawal a relapse occurred. On February 5 the same dosage was resumed and continued for 44 days altogether. No relapse then followed its cessation and the child made a complete recovery. The mastoids were not infected with yeasts.

In 1942 a case occurred for the particulars of which I am indebted to Lieutenant-Colonel J. M. Bonnin, Australian Army Medical Corps, who received them from Major E. B. Jones, Australian Army Medical Corps, then pathologist to the 2/6 Australian General Hospital, Jerusalem.

The patient was a soldier, aged thirty-five years, who had been ill for a few weeks. His clinical course was that of chronic meningitis. His main symptom was lethargy. There were no localizing signs. On August 13, 1942, cerebro-spinal fluid examination revealed a protein content of 95 milligrammes per hundred cubic centimetres, 161 lymphocytes per cubic millimetre and 15 neutrophile cells per cubic millimetre.

Yeast-like organisms, some of which showed budding, were recognized. On incubation at room temperature and at 37° C. *Torula histolytica* was grown. The organism was Gram-positive—approximately the size of a red cell. Cultures of the organism grown were sent to Cairo and Jerusalem. The Central Laboratories in Cairo reported a torula-like organism to be present. At the Hadassah Medical Organization animal inoculations were carried out on hamsters. Of the animals inoculated, some were killed, and some died eighteen days after inoculation. Torulosis was found to be widely spread throughout the organs. Another animal was inoculated with the urine of an infected animal, and developed widespread torulosis.

On August 18, 1942, the cerebro-spinal fluid was substantially the same as before. The protein content was 95 milligrammes per 100 cubic centimetres; the chloride content was 650 milligrammes per 100 cubic centimetres; the lymphocytes numbered 192, monocytes 18 and neutrophile cells 30 per cubic millimetre.

The relevant post-mortem appearances were as follows. When the calvarium was opened, the *dura mater* was found to be somewhat adherent over the vertex and right frontal lobe. On removal of the *dura*, it was noted that the cerebro-spinal fluid was scanty in amount. The cerebral vessels were not markedly injected. No evidence of meningitis was seen on the superior aspect of the brain, but over the frontal lobe, near the mid-line, two opaque nodular masses, approximately three millimetres in diameter, were present. On removal of the brain a small amount of greyish translucent granulation tissue was present just posterior to the optic chiasma. Extending over the pons and down towards the medulla, more of this granulation tissue was noted, and

on the right side, about the middle of the pons, a few millary opaque masses were noted.

Examination of the pharynx revealed no abnormality. Fairly dense pleural adhesions were present over the whole surface of the left lung. The right lung was free of pleural adhesions, but the two lower lobes of the lung were collapsed. On removal of the left lung, an almost hemispherical tumour, approximately six centimetres in diameter, was found on the vertebral margin of the lung just superior to the hilum. On incision of this tumour, it was seen to be composed of soft greyish jelly-like material. A Gram stain of material from the tumour showed the presence of *Torula histolytica*. No important morbid conditions were found elsewhere.

Cases Recorded in Australia.

The first case in which torula was recognized as the cause of disease occurred in 1915, but it was not until 1927 that Cleland described "A Case of Systemic Blastomycosis with Formation of a Myxomatous-looking Tumour-like Mass".⁽⁹⁾ In this case the infection was limited clinically to a large mass in the right iliac fossa. The organism seems to be *Torula histolytica* (see Figure II). Cultures were unsuccessful and inoculated animals remained healthy for a year.



FIGURE II.

Cleland's case of myxomatous mass ten centimetres in diameter in the right iliac fossa due to torula infection; drawings of torula cells. Those with large mucinous capsules had been treated with weak acid. (Vide THE MEDICAL JOURNAL OF AUSTRALIA, March 5, 1927.)

The first case in Australia of torula infection of the central nervous system in which diagnosis was made during the patient's life was that of Swift and Bull in Adelaide.⁽¹⁰⁾ Freeman writes: "Swift and Bull in Australia described the first case in which from the beginning there could be no doubt as to the etiological relationship of the parasite to the clinical symptomatology." The yeast was revealed in the cerebro-spinal fluid by means of contrast staining by indian ink. The appearance of the organisms in the cerebro-spinal fluid is shown in Figure III. The organisms are probably *Torula histolytica*, although there was an "attempt at mycelial formation" in cultures. It seems that these may be buds with elongated stalks.

Since then about seven more cases have been recorded in this country in the States of Queensland, New South Wales, Victoria and South Australia. Certainly some others have been recognized, but not reported.

In 1935 Sawers and Thompson reported a case and gave an excellent account of the pathogenic yeasts.

CASE HISTORIES.

Case I.

On November 5, 1934, a patient was referred to me by Dr. Souter, of Yankalilla, South Australia.

He was a pastoralist, aged fifty-two years, and had enjoyed good health until the beginning of August, 1934, when he began to suffer from severe headaches, mainly frontal, which recurred every day. At the end of September he had a bad cold in the head and chest. He spat up a large amount of sticky mucoid stuff. On October 14 he took to his bed and remained there. Towards the end of October he began to vomit at irregular intervals. The vomiting occurred every day and was independent of food. It was sudden without preceding nausea.

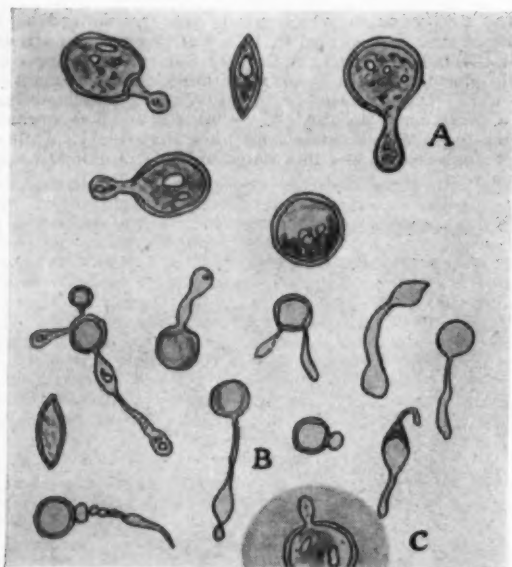


FIGURE III.

Swift and Bull's case; blastomycotic meningitis. A and C, in the cerebro-spinal fluid; B, in culture.

When examined at this time he was quite rational, but looked ill and haggard. His speech was normal. His thoracic and abdominal organs were normal on ordinary physical examination and the urine was normal. His cough had disappeared. The cranial nerves were not affected, except that intense double papilloedema was present. There were no disturbances of coordination or sensation. Flexion of the neck caused slight pain. The deep reflexes were not obtained, even with reinforcement. The abdominal reflexes were normal and the plantars flexor. The white blood cell count numbered 11,500 per cubic millimetre and the differential count was as follows:

Polymorphonuclear cells	84%
Eosinophile cells	nil
Basophile cells	1%
Lymphocytes	13%
Large mononuclear cells	2%

In a film, except for a slight variation in size and shape of the red cells, and the fact that the red cells were a little pale, no abnormality was detected.

Examination of the cerebro-spinal fluid revealed the presence of numerous white cells. Those seen were practically all lymphocytes with only one or two polymorphonuclear cells. Amongst the cells were numerous spherical objects varying in size from that of a lymphocyte to about twice its diameter. The bodies had a thick cell wall and resembled yeast cells. They stained also in a manner similar to yeast cells. One also was seen that appeared to have a bud from it. After incubation of a specimen for sixteen hours no culture was obtained. The globulin content of the cerebro-spinal fluid was greatly increased; sugar was present and the chloride content was 650 milligrammes per one hundred cubic centimetres.

The patient was admitted to a private hospital, where he remained until his death on January 16, 1935. During these ten weeks his condition steadily deteriorated.

He became increasingly helpless and during the last five or six weeks irrational. Repeated lumbar puncture relieved his intense headache.

On December 21 he had a general epileptic convulsion, and thereafter had two or three every week, whilst involuntary twitches of various muscle groups occurred frequently every day, lasting for a few minutes and passing off without a general convulsion. As time went on, the papilloedema was succeeded by optic atrophy, and during the last two or three weeks he was completely blind.

On January 6 it was noticed that he was slightly deaf. Rapidly the hearing diminished, so that a week later it was reported that deafness appeared complete. However, vestibular reaction to syringing was present on January 10.

During the patient's ten weeks' stay in hospital a fairly high degree of pyrexia was present with a large diurnal variation. The temperature reached 100° to 103° F. in the evenings and was subnormal in the mornings. The variation of the pulse accompanied the temperature—90 to 120 per minute. For the last three days he was or seemed to be comatose. The temperature charts are shown in Figure IV.

Treatment.

On November 9 double antrostomy was performed; a slight mucous discharge was drained which soon cleared up. Lumbar puncture was repeated every three or four days for the relief of headache whilst the patient was in hospital, ten or fifteen cubic centimetres of fluid escaping on each occasion.

Potassium iodide was given in doses of two grammes three times a day. Tryparsamide given intravenously and hexamine given by mouth were tried without any effect on the patient's condition. Deep X-ray treatment was applied to the brain and spine on several occasions with no beneficial result.

Laboratory Tests.

On November 6, 1934, Dr. Ray Hone examined the blood. The cytology and film showed no noteworthy departure from normal. The leucocytes numbered 11,500 per cubic millimetre, of which 84% were polymorphonuclear cells.

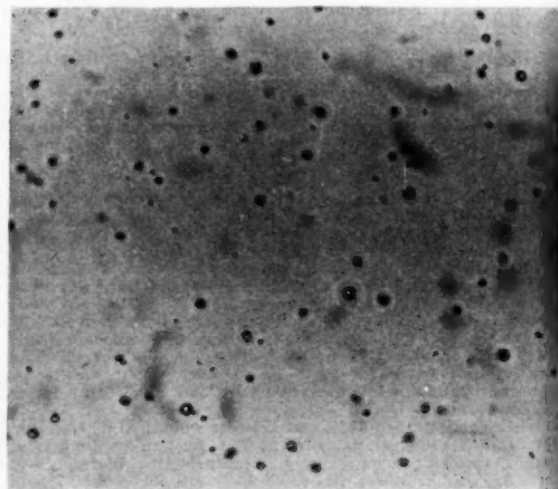


FIGURE V.

Case I. Numerous torula cells in the cerebro-spinal fluid (low power of the microscope). Capsules are shown as pale areolae.

The Cerebro-Spinal Fluid.

The cerebro-spinal fluid was examined by Dr. Ray Hone. The report submitted by him on November 13 is as follows: "To the naked eye the centrifuged fluid showed a colourless limpid supernatant layer, while at the bottom of the tube a dense white solid plug was seen. This plug consisted almost entirely of yeast cells. All specimens showed much the same characteristics" (see Figure V).

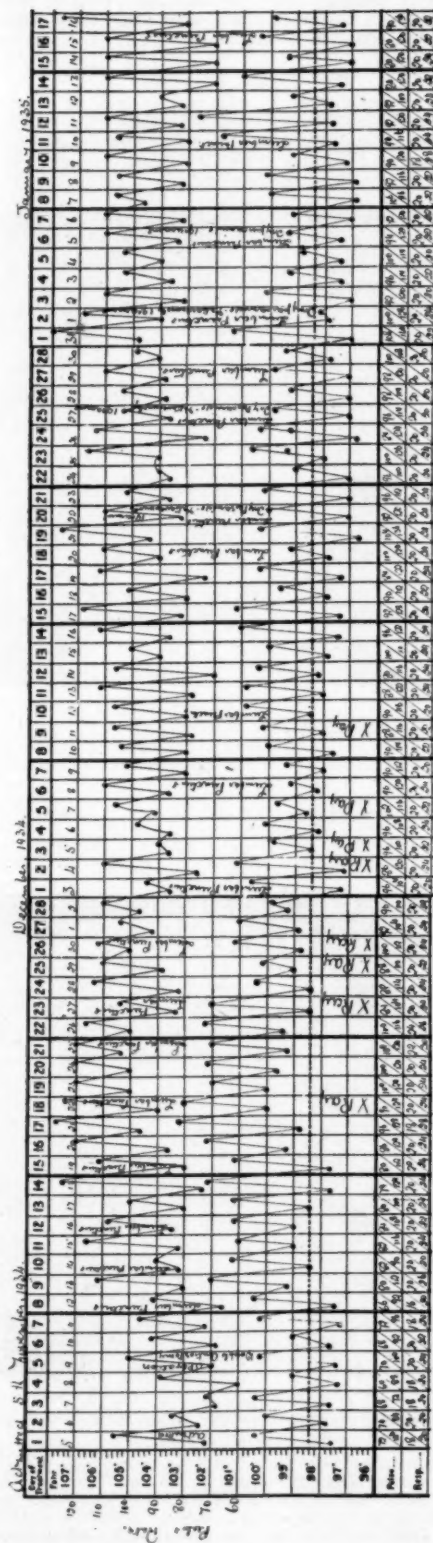


Figure IV.
Temperature chart for Case I.

Before passing to the pathological features of the case, I should say that after the patient's death his relatives informed me that he had been shot in the chest by a pistol at the age of seven. The bullet had not been recovered. The small scar was not seen.

Microbiological Investigations.—Microbiological investigations were carried out under the direction of Dr. L. Bull, then Director of the Government Laboratory of Pathology and Bacteriology, by Mr. S. Tee, who has prepared these notes.

The cerebro-spinal fluid when received showed a clear colourless supernatant fluid and a heavy, translucent sediment.

Under low power ($\times 100$), microscopic examination of an unstained wet preparation revealed numerous large, highly refractile bodies, varying in size from 5.0μ to 15.0μ , including budding forms with a capsule entirely surrounding the body. This capsule could be plainly seen under low-power examination (Figure V), but was not apparent under high-power ($\times 625$) lens (Figure VI).

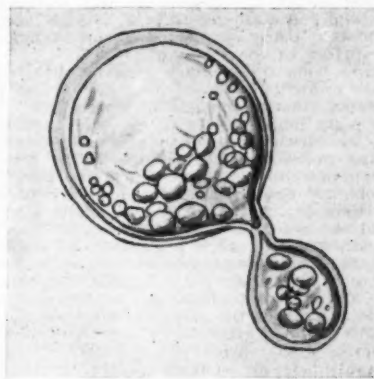


Figure VI.
Case I. Budding torula cells in the cerebro-spinal fluid (high power of the microscope). Capsule is not visible.

The results of laboratory investigations are summarized in the accompanying table.

Table Showing Results of Laboratory Investigations in Case I.

Observation.	Result.
Staining properties	Strongly Gram-positive. See pictures II and III of heat and formalin fixed and washed original material.
Media incubation	Incubated at 22°C . and 37°C . for 24 to 96 hours.
Incubation with Loeffler serum	Small clear colonies after 24 hours. Moist, coalescent within 48 hours. Thick, creamy, translucent in 96 hours. In cultures and subcultures.
Incubation with glucose broth	Moist, creamy growth at base only at 37°C . At 22°C . cultures less moist, flaky at the edges.
Fermentation with sugars, 14 to 30 days at 37°C .	Acid formation on dextrose only. No fermentation with other sugars.
Results of incubation with—	
Milk	Unchanged.
Gelatin	Surface cultures only, no liquefaction.
Simple agar	Fine yellow powdery growth.
Blood agar	No α or β hemolysis.
Anaerobic condition	Obligate aerobe—no growth in McIntosh and Filides' jar.

Specimens of deposit dried and fixed by heat could not be satisfactorily stained by Gram's method, but when the preparation was dried and treated for two minutes with 40% formaldehyde, followed by thorough washing in distilled water, perfectly stained specimens showing the capsule were obtained for examination under the oil-immersion objective (see Figure VII).

The examination of this and many subsequent specimens failed to reveal the presence of any bacteria or of any body cells.

Cultural Reactions.—Heavy inoculations of the sediment were made onto Löffler inspissated serum and glucose broth. On examination after twenty-four hours at 37° C. the Löffler serum slant showed minute colourless colonies extending up the slope to a height of two millimetres above the condensation fluid at the base of the slope. This was shown on microscopic examination to consist of strongly Gram-positive yeast bodies, which were much smaller and rather more oval in shape than the forms seen in the cerebro-spinal fluid.

Some of the large forms found in the deposit were seen in the culture films, and the daughter yeast cells, *in situ*, were clustered thickly round the circumference of the capsule. This growth phase is well demonstrated in Figure VIII.

When stained with Löffler's methylene blue, numerous vacuolations are observed in the culture forms.

Further incubation to forty-eight hours showed an increase in size of the colony to 0.5 millimetre in diameter, and after ninety-six hours at 37° C. the colonies had become large and moist, and had coalesced to form a thick, creamy, somewhat translucent mass, very moist in consistency, and raised from the surface of the medium.

Subcultures from the primary growths onto Löffler serum and glucose broth and incubated at 37° C. and 22° C. produced growths differing greatly in appearance.

In forty-eight hours at 37° C. the same abundant, moist, somewhat cream-coloured growth was obtained and the broth culture showed a marked amount of deposit with a clear supernatant fluid, this growth at the base of the tube being a constant characteristic when grown in fluid media, and at no time during the entire investigation was turbidity of the fluid observed, nor was pellicle growth seen.

The growth at 22° C., while being quite as vigorous as that at 37° C., differed in being less moist and distinctly yellow in appearance, and after several days' incubation, became quite dry, and tended to flake at the edges of the growth mass. This constituted the greatest difference to the cultures kept at 37° C., which after seven days still showed a moist and glistening appearance.

The organism is an obligate aerobe, surface and deep inoculations showing no growth when incubated in a McIntosh and Fildes jar.

Cultivation under aerobic conditions on simple nutrient agar and hormone agar at both 22° C. and 37° C. resulted in a fine, yellowish white powdery growth at both temperatures, the moist type of growth appearing only on coagulated serum. Cultivation on blood agar showed neither α nor β hemolysis.

Biochemical Reactions.—A complete series of carbohydrate media was inoculated and incubated for fourteen days at 37° C. Acid production was observed in dextrose. No other sugars were fermented, and milk was unchanged. Gelatine, after prolonged incubation, showed only surface growth and no liquefaction. The sugar media were incubated for thirty days, and although an abundant deposit of growth occurred in all tubes, no other sugars were fermented.

Animal Experiments.—Intraperitoneal and subcutaneous inoculations into guinea-pigs produced no lesions, and intravenous and intrathecal inoculation into rabbits gave negative results indicating that for these laboratory animals the organism is apparently non-pathogenic.

Resistance.—Specimens of cerebro-spinal fluid were kept at room temperature, which on occasions reached 90° F. and in full daylight for periods up to forty-four days, and were then seeded onto serum stabs, and in all cases vigorous growth was obtained, and from this growth subcultures were made.

In none of the primary cultures nor in subcultures was any attempt at mycelial formation observed, and it was impossible to reproduce on artificial media the large encapsulated form seen in the cerebro-spinal fluid.

This organism would appear to be very closely related to that described by Swift and Bull in the cerebro-spinal fluid in their case in 1916, the principal differences being that the present type grows more rapidly and does not attempt the formation of mycelia in primary cultures.

At my request, Dr. E. Britten Jones took some of the cerebro-spinal fluid from Case I to England in 1935, and it was given to Dr. Greenfield, pathologist at the National Hospital for Nervous Diseases, Queen's Square. Dr. Greenfield sent some of the fluid to the Lister Institute, where the yeast type was identified. It was found that the type was unusual and the Lister Institute had only one

similar strain among their type cultures. It is of interest that the strain was one that has also come from Adelaide and had been sent to the institute by Dr. H. Swift many years previously.

Post-Mortem Findings.

Post-mortem examination was carried out by Professor J. B. Cleland.

The brain to the naked eye showed some swelling of the structures at its base and a little gelatinous material in the pia-arachnoid meshes over the cerebellum. On close inspection

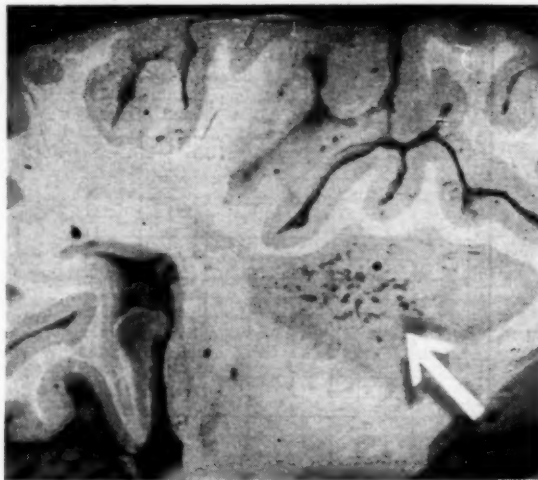


FIGURE IX.
Case I. "White-anting" of the left parietal cortex and basal nuclei by torula cells accompanying the vessels, the "white-anting" exaggerated by the development of the mucinous capsules.

minute little sago-like specks up to the size of a pin's head could be seen in the cortex. These were irregularly distributed, but often occurred in clusters in the grey matter, occasionally extending to the underlying white matter.



FIGURE X.
Case I. Torula infiltration of the right parietal cortex and basal nuclei.

The putamen on both sides, but especially the right, was honeycombed by slightly larger mucinous cavities. These were less numerous in the caudate nuclei and optic thalami. The appearances resembled on a minute scale the tunnelling of "white ants" (termites). (See Figures IX and X.)

In the middle lobe of the right lung was a nodular myxomatous-looking mass one inch (2.5 centimetres) in diameter. In the lower lobe immediately below this a revolver bullet of the calibre 0.22 was found. There was some diffuse pneumonia in both lower lobes (Figure XI).



FIGURE XI.

Case I. Lung; naked-eye photograph. Just below the arrow is a myxomatous-looking mass in the middle lobe due to torula cells. Below this again is the embedded 0.22 bullet in the lowest lobe.

Microscopic examination showed yeast-like bodies surrounded by mucinous capsules 9μ to 13μ in size (including the capsule). These diffusely infiltrated the meshes of the pia-arachnoid and accompanied this along the course of the blood vessels into the substance of the brain. As these cells multiplied, they became surrounded by extensive mucinous capsules, and this mucinous material simply shoved aside adjacent structures. In this way small spaces containing mucinous material were formed where the pia penetrated into the surface of the brain (Figures XII and XIII). In the putamen some of the spaces thus produced were from 0.4 by 0.16 millimetre to 1.0 by 0.3 millimetre in size. These yeast-like cells were also numerous in the sheath of the optic nerve (Figure XIV). In the auditory nerve the yeast cells infiltrated between the nerve bundles (Figure XV). Masses of yeast cells were responsible for the nodule in the lung. (See Figure XVI—the bronchiole with torula cells are shown above and emphysema is shown below. The magnification is $\times 30$.)

The reaction to these torula cells appears to have been very slight. In some places there was no cellular response. In the meninges there was some increase of reticulo-endothelial cells; the nuclei of cells of this type were sometimes seen surrounding torula cells, and occasionally a torula was seen in a large granular cell with multiple nuclei, but typical foreign body giant cells were not noted.

How did infection originate? The mature cells are too large, being mostly over 9μ in size, to be ingested by macrophages and so carried into the stroma of the lungs or elsewhere. Moreover, the mucin outside the capsule would protect the torula from ingestion. On the other hand early budding stages might be separated when still quite small and so possibly be ingested by macrophages. If the torula does not gain access to the tissue spaces by being carried in by phagocytes, we are left with the alternative that it must be introduced by a wound of some nature, though it is possible that a yeast cell might be inhaled, might enter eventually an alveolus and be unable to escape owing to its mucinous capsule, and that it then might multiply, and eventually the mucin might distend and burst the alveolar walls, and so the yeast cells might enter the stroma of the lung.

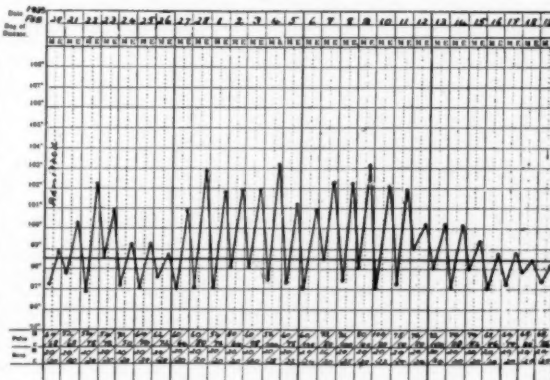
The last mentioned, perhaps, is the best explanation of the origin of this infection. This would assume that the lung lesion was the primary lesion. It is remotely possible, of course, that the bullet which entered the lung when the patient was a child might have carried with it a torula which was left behind in its trail and gradually multiplied to produce the mass in the lung. This would presume an infection of many years' standing with the terminal accidental escape of yeast cells into the circulation and their arrest in the capillaries of the membranes of the brain and thence their extension to the lepto-meninges. However the organism entered the body, this route of infection to the brain must surely have been the one which was followed.

Case II.

P.S.H., a pastoralist, aged twenty-nine years, had suffered occasionally from dyspeptic symptoms which ceased after a retrocaecal adherent appendix was removed in August, 1936. Otherwise he was a healthy active person.

The first significant incident in his history occurred early in February, 1939. He began to have frequency of micturition with urethral scalding. The urine was turbid and acid. It contained pus cells and a few casts. The specific gravity was 1.015. No albumin or sugar was present.

He was admitted to hospital and remained there from February 20 until March 29. For twenty-six days from the time of admission pyrexia was present. He had frequent sweats and muscular pains and backache, but no other noteworthy symptoms. The evening temperature ranged from 99° to 103° F., usually about 101° F. The morning temperature was always normal or below (see Figure XVII). The frequency of micturition gradually abated and the urine was clear after about a fortnight. No microorganisms were demonstrated in it at any time either by direct examination of the deposit or by culture.

FIGURE XVII.
Temperature chart, Case II.

Relevant laboratory tests showed that the blood failed to agglutinate *Bacillus paratyphosus* A and B, *Bacillus proteus* X19 and *Micrococcus melitensis*. Attempts at blood culture carried out on two occasions were fruitless. Red blood cells on March 10, 1944, numbered 3,700,000 per cubic millimetre. The haemoglobin value was 65%. Leucocytes numbered 3,000 per cubic millimetre on three occasions and the numbers of all varieties were equally reduced.

This illness terminated without our discovering the nature of the infective agent. The patient left hospital apparently well on March 29. He gained strength and weight and his colour improved.

On May 1 he again consulted me. He complained of a severe attack of migraine, to which he was subject. He had a constant headache with a sense of fullness and tension which had persisted for three days before I saw him. He was nauseated all this time and had vomited on the night of April 30. He entered hospital on May 2. For the first week his condition changed very little. He looked pale and ill. He suffered from headache to a varying degree. It sometimes disappeared and was never very severe. He slept fairly well with mild sedatives. Nausea was frequently complained of and he vomited quite suddenly on several

occasions. Profuse sweating occurred from time to time. During his first week in hospital his temperature was above 99° on only one day. His pulse rate varied between 46 and 66. On May 10 he complained of pain in the back and stiffness in the neck. At the same time he had a sore throat. His fauces, uvula and pharyngeal wall were swollen and congested. The temperature rose at this time to 101° F. on May 15. Thereafter until forty-eight hours before death pyrexia persisted. His mind was clear throughout, though during the last week he was somnolent, but when roused he answered intelligently and had insight into his condition (see Figure XVIII).

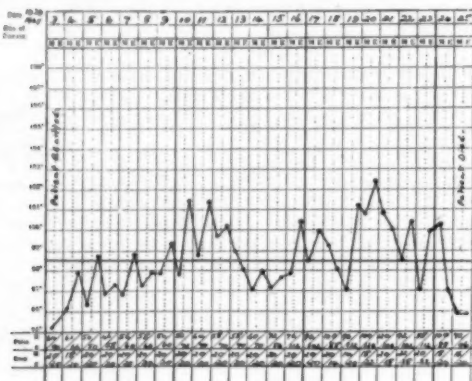


FIGURE XVIII.
Temperature chart, Case II.

The blood was examined on May 13, 1939. The leucocytes numbered 5,500 per cubic millimetre. The differential count was as follows:

Polymorphonuclear cells	75%
Lymphocytes	12%
Monocytes	12%
Eosinophile cells	1%

The serum failed to produce agglutination with *Bacillus typhosus* (H and O suspensions), *Bacillus paratyphosus* B, *Bacillus proteus* OX19 and *Brucella abortus*.

On the tenth day of the patient's stay in hospital papilloedema (+ 4 diopters) was found in the left eye. There were two hæmorrhages into the disk. The right fundus was normal. At this time the abdominal reflexes could not be obtained. The deep reflexes were very sluggish, the plantar reflexes were extensor and Kernig's sign was present. Lumbar puncture was performed on the nineteenth day, and was repeated daily.

The Cerebro-Spinal Fluid.

The cerebro-spinal fluid was at a pressure of 300 millimetres of water at first, but later showed lower pressures 170 to 250 millimetres.

The fluid was not definitely tinted, but was turbid. A few creamy flakes were present in the fresh fluid and a creamy deposit formed on standing, but no clot formed.

After centrifugalization it appeared quite clear with a slightly yellowish tinge. At the bottom of the tube was a pearly white plug. Globulin was found to be present in excess. The protein content was 120 milligrammes per hundred cubic centimetres. The chloride content was 660 milligrammes per hundred cubic centimetres. One lymphocyte was present in each cubic millimetre.

Examination of the deposit revealed many colourless particulate bodies, spheroidal in shape and varying in size from that of an erythrocyte to one a quarter of that size. These bodies were Gram-positive and one or two were seen to be budding.

On May 20, 1939 (one day after the collection of the fluid), no culture had been grown.

The only fluid available for chloride estimation was the supernatant fluid obtained after prolonged centrifugalization. This had been too long in contact with the infecting organism for accurate figures to be obtained.

A culture of torula was obtained from a specimen of blood removed on May 16.

Sulphapyridine was given in full doses. A concentration of five milligrammes per hundred cubic centimetres was obtained in the cerebro-spinal fluid on May 26, but the patient's condition rapidly became worse. The findings on three previous examinations to determine the concentration of sulphapyridine were as follows: on May 22, 1.7 milligrammes per centum; on May 23, 3.6 milligrammes per centum (five yeast cells were found per high-power field in one specimen and seven specimens in another); on May 24, 4.9 milligrammes per centum.

For forty-eight hours before he died the patient was in a state of peripheral circulatory collapse with rapid feeble pulse and cold extremities.

Dr. Eugene McLaughlin examined several specimens of cerebro-spinal fluid removed on different occasions. The significant observations common to them all were cytological and biochemical. In regard to the former, the leucocytes numbered about 25 per cubic millimetre, and polymorphonuclear cells about four or five per cubic millimetre. Yeast cells were abundant and some budding forms were seen. On the biochemical side the protein content was 140 milligrammes per centum, globulin was present in excess, only a trace of glucose was found and the chloride content was 650 to 660 milligrammes per centum.

The yeast cultures were sent to England for full investigation of cultural reactions and comparison with type cultures. Unfortunately war prevented this being carried out.

Animal Inoculations.

Animal inoculations showed the yeast to be pathogenic for rabbits. Pleurisy with fibrosis and obliteration of pleural spaces followed inoculation into the pleura. Intravenous inoculation caused death three weeks later. The lungs showed fibrosis and yeast infiltration.

Post-Mortem Findings.

Dr. Thiersch, who reported on the post-mortem findings, said that the main features of the case were the rapid course, general dissemination and involvement of most of the organs in the torula infection. The resemblance to tuberculosis in dissemination and structure of some of the lesions was striking.



FIGURE XIX.
Left lung, subpleural focus.

Lesions were found in the left lung which might have been the focus of primary infection. From here the dissemination in the lymph and, later, blood stream took place involving broncho-pulmonary lymph nodes, then both lungs with bronchopneumonia and pleurisy, the lymph nodes of the bifurcation of the trachea and the paratracheal lymph nodes, the mesenteric lymph nodes and also retroperitoneally the paraaortic lymph nodes.

Further, the spleen, left kidney, adrenals, pancreas and prostate were involved as evidence of the hæmatogenous

dissemination, the actual torula lesions, particularly in spleen, prostate, adrenal and pancreas, resembling tuberculomata to an extraordinary degree. Finally a leptomenigitis involving the pia and arachnoid and chorioid plexus developed, bringing about the death of the patient.

A number of photographs were taken to demonstrate the lesions and their distribution. As, however, space is limited, only a small number of prints were selected, showing the similarity to tuberculosis and the specificity of the torula infection.



FIGURE XX.
Case II. Right lung with bronchopneumonia in the upper lobe.

Figure XIX shows the left lung with subpleural focus (? primary infection) of the lung. Further small areas of bronchopneumonia and small patches of pleurisy are visible.

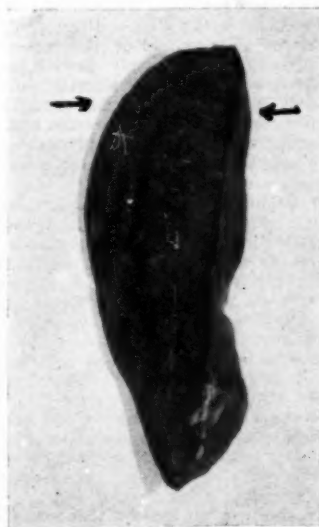


FIGURE XXIII.
Spleen (macroscopic): tubercular nodules.

Figure XX shows the extensive torula bronchopneumonia in the upper lobe and upper parts of the lower lobe of the right lung, patchy pleurisy and torula lymphangitis.

Figure XXI shows the subpleural focus of the left lower lobe. An area of necrosis without much inflammatory reaction at the edge of the normal lung is visible. An area of necrosis with yeast is marked "a" and lung tissue is marked "b". The magnification is $\times 75$.

Figure XXII shows the actual area of necrosis, filled with debris and numerous yeast bodies, many of which are surrounded by a halo. The faint outline of a smaller bronchus is still visible in the necrotic area. The magnification is $\times 210$.

Figure XXIII shows the spleen macroscopically. The irregular outline of the granulomata are best visible at one pole, but were scattered throughout the organ. The spleen was only slightly enlarged and firmer than normal. The granulomata were greyish in colour and of the size of beans.

Figure XXIV shows a microscopic view of the lesion in the spleen. Around an area of central necrosis (marked "a"), granulations are found, which closely resemble tuberculosis. Epithelioid cell proliferations are marked "b", and numerous Langhans giant cells and lymphocytic infiltrations forming tuberculoid structures are marked "c". The foci often started in the lymph follicles and extend from here directly or by confluence. On close examination no tubercle bacilli but numerous torula bodies were found in these granulations, the majority intracellularly in the giant cells. The magnification is $\times 75$.

Figure XXV shows the prostate and bladder. An area of extensive necrosis resembling a tuberculoma in the middle of the prostate was found. On microscopic examination this area consisted of conglomerated tuberculoid structures with an extensive central necrosis. The edges were lined with epithelioid cell proliferations and Langhans giant cells. Both granulations and necrotic centre contained numerous yeast bodies.



FIGURE XXV.
Prostate (macroscopic): (a) yeast granuloma; (b) bladder.

Figure XXVI shows a microscopic picture of the edge of the granulomatous areas in the prostate: epithelioid cells, Langhans giant cells and lymphocytic infiltration with fibrosis have replaced the original prostatic structure. A yeast granuloma with giant cells is marked "a"; prostate concretions are marked "b". The magnification is $\times 75$.

Figure XXVII shows the enlarged mesenteric lymph nodules in the centre, the liver and stomach on one side.

Figure XXVIII shows the enlarged paraaortic lymph nodules forming a conglomeration surrounding the aorta; again, liver and stomach are seen in the specimen.

Figure XXIX. The microscopic picture of the affected lymph nodules is quite unlike a tuberculous lymphadenitis. The consistency of the lymph nodules was rubbery and firm, and a general hyalinization has taken place. The structure is completely destroyed, all the lymph follicles have disappeared, and only a few small arteries and strands of connective tissue remain.

To the naked eye, the brain was not affected. There was, however, much leptomenigitis, with a gelatinous thickening and exudate at the base of the brain following the arachnoid into the intergyral spaces. The chorioid plexus was also involved and thickened.

Figure XXX. Microscopically, the arachnoid and *pia mater* are both thickened. The subarachnoid space is filled with a serous exudate containing numerous torula bodies. Both pia and arachnoids show infiltration with endothelial cells, often enclosing yeast bodies surrounded by a clear halo. The letter "a" marks a yeast granuloma; "b" marks brain tissue. The magnification is $\times 30$.



FIGURE XXVII.
Paraaortic lymph nodes: (a) lymph nodules; (b) liver;
(c) aorta.

Figure XXXI shows an artery of the arachnoid, marked "a", and the subarachnoid space. The arachnoid, marked "b", is severely infiltrated with endothelial cells containing numerous yeast bodies, then follows an area of serous exudate, marked "c", with numerous free torula organisms, which on the other side meets the thickened *pia mater*, again infiltrated by endothelial cells with numerous torula bodies, marked "d".



FIGURE XXVIII.
Case II. Mesenteric lymph nodes: (a) lymph
nodules; (b) liver.

Figure XXXII shows intracellular torula bodies with large clear halos, phagocytized in endothelial cells in the subarachnoid space. The magnification is $\times 500$.

Commentary.

These two cases showed striking differences. The first man was ill for several months with steadily ingravescent symptoms. The pathological examination showed that the deposits of yeast cells caused but little reaction in the surrounding tissues. Further, the yeasts failed to reproduce the disease in the animals inoculated.

The illness of the second patient was of considerably shorter duration and was divided into two distinct phases. The final meningitic stage was rapidly fatal. At its height cryptococci were cultivated from the blood. The tissues showed very active local defensive reaction with proliferation of the reticulo-endothelium and giant cell formation, in parts resembling tuberculosis, in the lymph glands not unlike lymphadenoma. The animals inoculated all died. Their organs showed much subacute inflammatory reaction.

Unfortunately in the second case the cultural and other characteristics of the torulae were not studied, but from the very different reactions in the tissues of the hosts it seems likely that the infective agents were of different species or varieties. The records of other cases confirm this view.

Mode of Entry.

Explanation has been difficult in many recorded cases, but in others it is easier to understand. In several instances yeast cells have infested tuberculous cavities in the lungs and thence invaded the blood stream. In Greenfield's case the cells may have entered the circulation from an infected wound. In others no such route has been discovered. Apparently no authentic examples of entry via the upper air passages have been recorded, and most observers have agreed that it is from the lower respiratory tract that invasion usually occurs. In my two cases, especially the first, the lungs seem to have been the site of the earliest lesions. It may be remarked that both men were pastoralists. Possibly the yeast cells are conveyed in the dust of dried herbage or from that of drafting yards. Torulae seem to be only accidentally pathogenic, and without our knowing more about their normal habitat in nature, it is impossible to say what material is most likely to be infected by them.

Pathology.

Freeman,¹⁰ discussing the lesions of the brain found at necropsy, divides them into three main types: (i) Diffuse or granulomatous meningitis. (ii) Small granulomata or cysts in the cortex, apparently extending from the surface of the brain along the course of the vessels into the cerebral parenchyma. (iii) Deeply placed lesions, sometimes solid, oftener cystic, occasionally diffuse, lying chiefly in the grey matter of the basal ganglia, but sometimes in the white matter of both cerebrum and cerebellum. These are assumed to be embolic in origin.

The first two types of lesions were seen in both cases. The third was present only in Case I.

In regard to tissues other than the brain, the degree of reaction of the part to the implantation and multiplication of the yeast cells shows remarkable individual differences. Greenfield, Martin and Moore¹¹ suggest that this probably depends on difference in virulence in various strains, and, further, that cases in different parts of the world are not all due to the same organism. Duration does not account for this difference, since there was much more hyperplasia in the more acute of my two cases than in the more long-standing one. Greenfield and his co-workers quote Mitchell as saying that "large lymphatic tumours are often produced by invasion of the lymph nodes and it is probable that the lymphatic system offers the most active defence against a general spread of the infection".

The massive lymph glands in the second case are an example of this; the prostate, spleen and lungs also showed a granulomatous reaction.

Symptomatology.

The duration of cases of torulosis of the central nervous system culled from the literature varies from three to four weeks to two and a half years. Most patients are afebrile.

ILLUSTRATIONS TO THE ARTICLE BY C. T. CHAMPION DE CRESPIGNY.

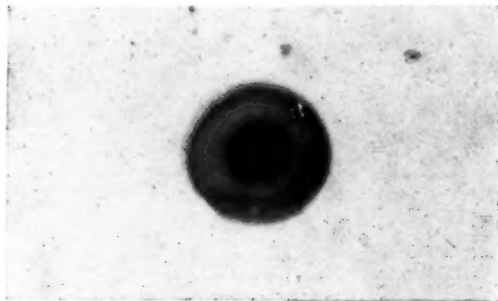


FIGURE VII.

Case I. Stained torula cells from the cerebro-spinal fluid, showing capsule. (Oil immersion lens.)

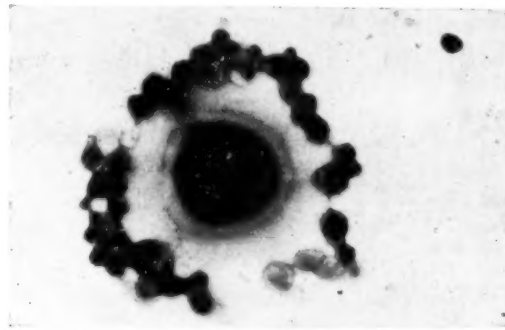


FIGURE VIII.

Case I. Culture showing daughter yeast cells clustered round the capsule of the parent cell. Note the double contour of the cell and the mucinous envelope beyond this. (Oil immersion lens.)

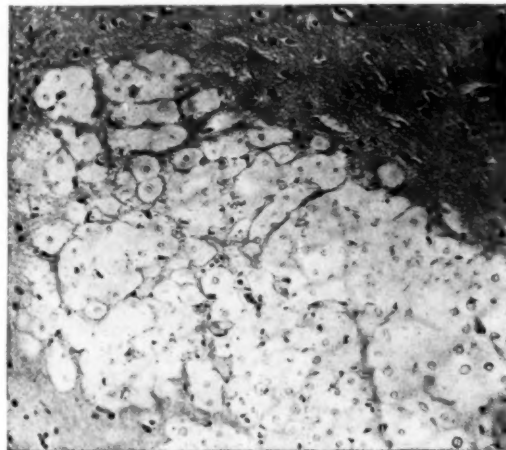


FIGURE XII.

Case I. Torula cells in mucinous capsules accompanying pial extensions round vessels in the cortex.

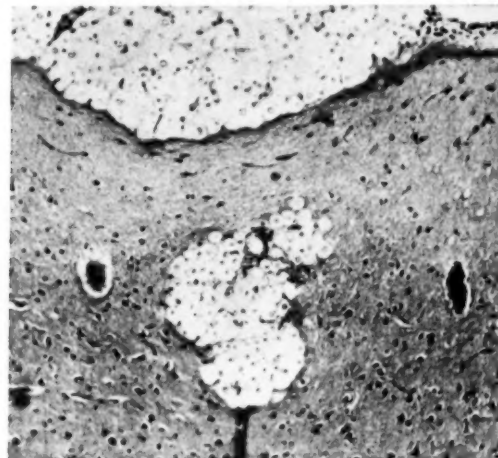


FIGURE XIII.

Case I. Left parietal cortex. Infiltration of the pia (above) by torula cells, which have accompanied vessels into the cortex.

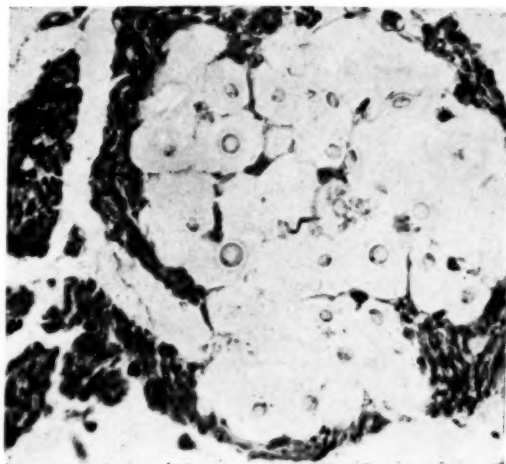


FIGURE XIV.

Case I. Optic nerve (below). Sheath infiltrated with torula cells. ($\times 60$.)

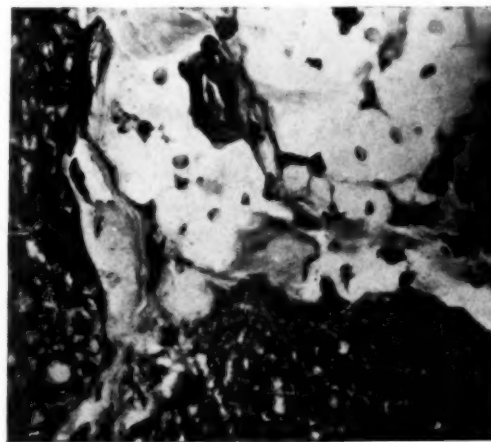


FIGURE XV.

Case I. Auditory nerve. Torula infiltration between bundles. ($\times 60$.)

ILLUSTRATIONS TO THE ARTICLE BY C. T. CHAMPION DE CRESPIGNY.

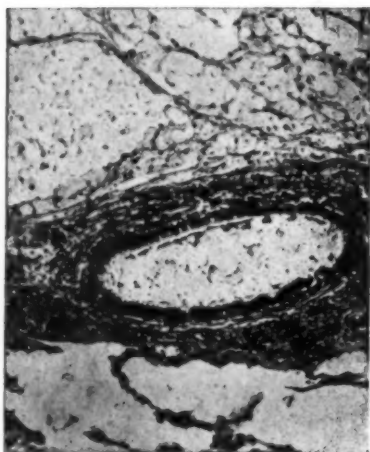


FIGURE XVI: Case I.



FIGURE XXI.

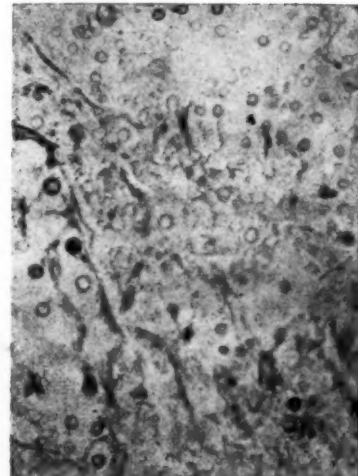


FIGURE XXII.

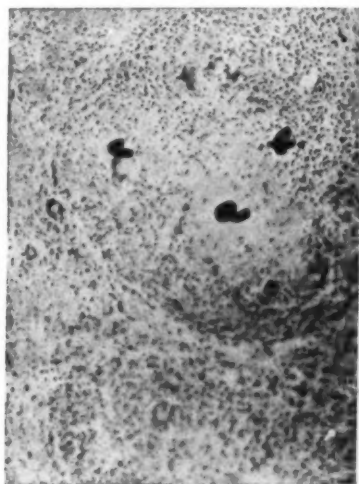


FIGURE XXIV.

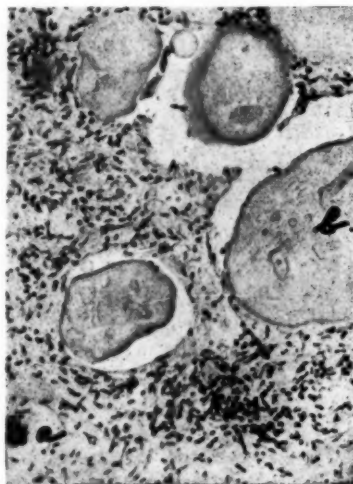


FIGURE XXVI.



FIGURE XXIX.

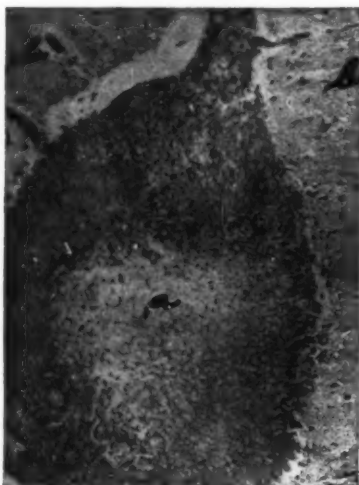


FIGURE XXX.

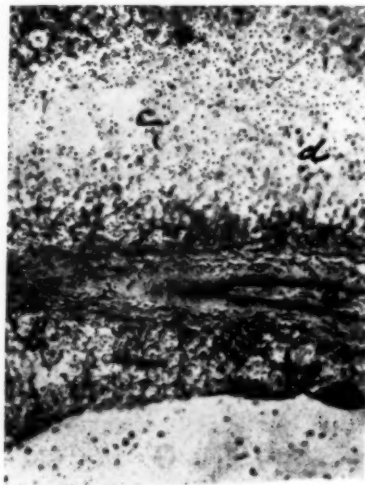


FIGURE XXXI.

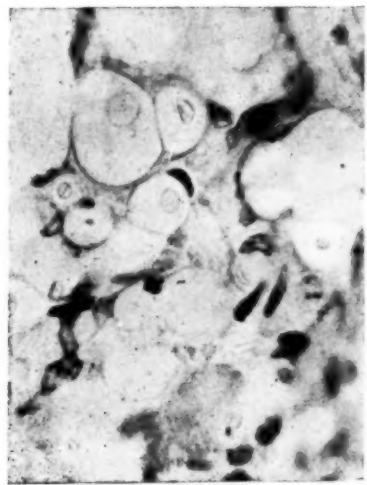


FIGURE XXXII.

Both mine showed active pyrexia. The second exhibited two distinct phases in his illness. A febrile attack lasted for about a month and was followed by apparent recovery. After another few weeks in which he seemed quite well the patient became acutely ill with meningitis to which he soon succumbed. I believe that the first pyrexial episode marked the stage of visceral invasion, but that the brain and meninges so far escaped. The vigorous local reaction walled off the torulae and sealed up the source of toxæmia. Then came an overwhelming invasion of the blood stream (we recovered torulae by blood culture), the meninges were attacked, and the second acute final stage ensued.

It is well to remember that great enlargement of the superficial and deep lymph glands may be caused by torular infection. On several occasions such cases have been mistaken for Hodgkin's disease.

The nodules in the lungs might have given rise to much difficulty in diagnosis had the second patient been examined by X rays. They could have been mistaken for secondary new growths or sarcoidosis.

All writers on the subject agree that headache, persistent and often intense, is the most constant feature of the disease. This is readily to be understood from the high intracranial pressure that it produces.

This high pressure I believe to be caused by yeast cells blocking the routes of exit of the cerebro-spinal fluid via the Pacchionian bodies from the subarachnoid space into the venous sinuses, but I have not proved this in the cases considered.

Meningeal symptoms are nearly always present. Often nerve palsies, diplopia, blindness and even deafness occur. In the absence of pyrexia, intracranial tumour is a likely diagnosis. When pyrexia is present, tuberculous meningitis is most probably suspected, especially if already the patient is suffering from pulmonary tuberculosis. The presence of enlarged lymph glands resembling Hodgkin's disease may make one suspicious, but it is only the discovery of torulae in the cerebro-spinal fluid that will confirm the diagnosis.

The Cerebro-Spinal Fluid.

In some recorded cases torulae were stated not to be present in the earlier specimens of cerebro-spinal fluid. When they have their characteristic double contour and refractility, variation in size and the presence of budding make them unmistakable objects. Dr. Bull's use of indian ink should be remembered. Torulae are easy to cultivate, and even if absent in the films, cultures will give the diagnosis.

The chloride content is usually in the region of 650 milligrammes per hundred cubic centimetres. Glucose is present in smaller quantity than normal or may be absent. Pleocytosis is variable. Lymphocytes predominate. They may be in numbers from twenty per cubic millimetre to a hundred or more, dependent upon the meningeal reaction.

The appearance of the fluid was very striking in both cases, especially the first. After spinning in the centrifuge the supernatant fluid may be clear and colourless or slightly yellowish, while at the bottom of the tube is a small solid plug, white like plaster of Paris.

Treatment.

In the first case iodide of potassium, "Stovarsol", hexamine and deep X-ray therapy were all used without effect. In the second a concentration of sulphapyridine up to five milligrammes per hundred cubic centimetres of cerebro-spinal fluid did no good. However, the American case of successful treatment with sulphadiazine leads one to hope that Freeman's gloomy statement that the disease is always fatal may not be true in the future.

ACKNOWLEDGEMENTS.

Finally, I must acknowledge the generous assistance which I have received in the preparation of this lecture from Professor J. B. Cleland, Dr. Thiersch, Mr. Tees and Mr. E. Rogers, and from the radiological department of the Royal Adelaide Hospital.

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OPERATION FOR ACUTE MASTOIDITIS.

By WALTER CROSSE, F.R.C.S. (Edinburgh), F.R.A.C.S.,

AND

L. T. JOBBINS, M.B., Ch.M.,
Brisbane.

IN view of the recent articles and correspondence in the journal on the Schwartz mastoid operation and the shortening of the post-operative treatment, this report of the following series of ten cases may be of interest.

The treatment was as follows. A simple mastoidectomy was performed in each case. Several of the mastoids were of the large pneumatic cell type. All cells were exposed and the diseased mucosa was thoroughly removed. Particular attention was paid to those areas in which cells might be overlooked, that is, tip cells, posterior meatal wall cells, cells running into the roof of the zygoma and those over the lateral sinus and postero-superior to the lateral sinus bend. At the completion of the excavation the wound was thoroughly syringed to remove all debris and spicules of bone, dried, and then the whole area dusted with "Thiazamide Sodium". A small curette full of the same crystals was dropped into the meatus and a plain gauze wick was inserted. The wound was completely closed with three to four vertical mattress sutures and a half-inch selvedge gauze drain was placed in the lower end of the wound. The whole area was then covered with a large sterile pad. The patients were given sulphadiazine for periods varying from five to seven days. The wick in the external auditory canal was removed in forty-eight hours, and if not dry a fresh wick was inserted. The sutures and gauze drain in the lower end of the wound were removed on the fourth day.

In all but two cases the ears were dry and the wounds healed within a week following operation. In the two cases referred to, there was some serous discharge from the lower end of the wound for over a week, but both

wounds were completely healed within a fortnight. The hearing has been good in all cases and no sign of any depression post-aurally is as yet visible.

The "Thiazamide Sodium" crystals (anhydrous sodium sulphathiazole) are put up by May and Baker in small glass bottles each containing five grammes of crystals. The crystals are sterile and are hermetically sealed. We have suggested to the company's representative that a smaller pack of about two grammes should be put up, as re-sterilizing in an autoclave causes the crystals to clump.

Acknowledgements.

This treatment was suggested to us by Captain Snow, of the United States Army Medical Corps, to whom we acknowledge our indebtedness.

Reports of Cases.

UNUSUAL COMPLICATION IN PROTRACTED INSULIN SHOCK TREATMENT.

By Z. WECHSLER, M.D.,
Heathcote Reception Home, Perth, Western
Australia.

PROTRACTED insulin shock treatment has been recommended by Kraulis⁽¹⁾ in 1938 for those chronic schizophrenics with whom previous shock therapy has been unsuccessful. Under this treatment patients are left in hypoglycemic coma for a period of from ten to twelve hours instead of one to one and a half hours as in the original Sakel's⁽²⁾ method. To reduce danger, at the beginning of the coma, usually from the third hour of hypoglycemia onwards, small doses of sugar are given by means of a nasal tube. The shock is interrupted by a large dose of glucose.

Of 22 patients treated by Kraulis by this method, five recovered completely, five were discharged from hospital "improved" and the condition of eleven remained unchanged. In view of the completely unfavourable prognosis of the cases reported, the results are rather encouraging, though the method involves considerable risk.

Protracted comas of from three to fifteen hours have been also advocated by Billig and Sullivan.⁽³⁾

A forty-five years old female patient, suffering from paraphrenia and undergoing this type of treatment in this hospital, developed recently an unusually long irreversibility lasting nearly eight weeks. After six months the clinical picture is that of a profound organic dementia.

D.R., mother of four children, gradually developed about three years ago a delusional state with fixed persecutory ideas and auditory hallucinations. Because of her antisocial conduct she had to be admitted on two occasions to this hospital.

Convulsive shock treatment and prolonged narcosis ("Medinal"-Luminal) had no effect. In view of the poor prognosis, it was decided to apply protracted insulin shock treatment. Her physical condition was excellent in every respect. The relatives were told of the risks involved.

An initial dose of 25 units of insulin was given and this was gradually increased by 15 units daily until a hypoglycemic coma was produced on the eighth day, with a dose of 130 units. It was intended to extend the coma to ten hours, if possible. Small doses of sugar (1-0 gramme) and saline solution were given every hour with a nasal tube. The patient awoke spontaneously after four hours and forty minutes.

On the ninth day the procedure was repeated, but, to enable the patient to remain in coma longer than on the previous day, the amount of sugar, given hourly, was reduced to half (0.5 gramme). After three hours and twenty-five minutes a hypoglycemic coma was induced, and this was extended to ten hours, as the patient was not displaying any symptoms which would necessitate an early termination of the coma. To interrupt the coma a large dose of sugar (200.0 grammes) was given, but the patient failed to regain consciousness. In spite of intravenous injection of glucose, vitamin B₁, sodium chloride (hypertonic solution), "Tetrazol", adrenaline given hypodermically and inhalation of "Carbogen" the coma became irreversible. The patient's condition became alarming and her name was placed on the danger list. A blood sugar test taken next day gave a

reading of 108 milligrammes per centum. The leucocytes numbered 10,800 per cubic millimetre. During the following days she developed signs of bronchitis and circulatory failure. Her temperature went up to 101.2° F. and her pulse rate to 132. She was given "M & B 693" for several days and cardiac stimulation every two hours. In due course she became increasingly restless; the movements became of a chorea-athetoid type. Abnormal associated movements were particularly marked in the face. Her facial expression became "grimacing" with peculiar tightening and screwing of the lips, accompanied by lingual licking movements. The pupils reacted to light and the reflexes were normal. No Babinski sign was elicited and the limbs were hypotonic rather than rigid. She had to be fed artificially and developed incontinence of urine and faeces.

After fifty-seven days she began to take food, and on the following day she appeared to understand questions, but was unable to answer them. Her speech, first distorted beyond recognition, due to verbigeration, gradually became more normal, though with marked loss of memory for words (amnestic aphasia).

At present, after six months, her physical condition is reasonably good. Her facial expression is somewhat flattened. She is disoriented as to time and place and her memory is very defective with marked confabulatory tendencies. She cannot tell the number of her children or their ages, but is able to recognize them. She is dull and indolent; she has little initiative and does not seem to worry about what is going to happen to her. She is highly suggestible, ready to obey simple commands, and on the whole easy to nurse. She has lost her hallucinations and does not seem to entertain her previous persecutory ideas. Her primary psychotic condition has subsided, but a state of organic dementia has developed, obviously as a result of the prolonged irreversibility of the coma. The administration of glucose, adrenaline, vitamin B, "Carbogen" and "Tetrazol" as advocated by various authors was completely ineffectual. Her survival during this critical period was due mainly to excellent attention on the part of the nursing staff.

Acknowledgement.

I am indebted to Dr. F. M. G. Prendergast, Acting Inspector-General of the Department of Mental Hospitals, for his encouragement and advice during the treatment of this patient and his permission to publish this report.

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AN ACCOUNT OF A GUNSHOT WOUND OF THE ABDOMEN.

By LOWEN A. HARDY,
Surgeon Lieutenant-Commander, Royal Australian
Navy.

D.S. was admitted to hospital at 4.15 a.m. on July 16, 1944, with a history of having been accidentally shot in the abdomen with a .303 inch bullet. The bullet entered the abdomen about one inch below and to the left of the umbilicus, and passed diagonally upwards and to the right and emerged from the body just above the costal margin about the mid-axillary line.

Examination revealed bullet wounds in the positions indicated, with no powder marks evident, indicating that the bullet had been fired some little distance away, rigidity of the abdominal wall, and slight dullness in both flanks. The patient was not unduly shocked and the pulse was strong, not unduly rapid; the systolic blood pressure was 150 and the diastolic pressure 80 millimetres of mercury. The heart sounds were clear. An unusual feature of the case was that the entrance wound was much larger than the exit wound.

Laparotomy was carried out by means of an upper right paramedian incision, under "open ether" anaesthesia. The abdomen contained a large quantity of free blood; the upper

loops of the small intestine were found to be perforated in eight places, with rents in the mesentery in four places, from which hemorrhage was very free. The perforations in the intestines were closed by a purse-string suture and the rents in the mesentery closed by the method described in Hamilton Bailey's "Emergency Surgery". Resection was not carried out as it was considered that the affected loops had a good chance of viability and that the patient's condition did not warrant such a lengthy procedure.

Peritoneal toilet was carried out, sulphanilamide powder being used; the abdomen was closed in layers with drainage in the lower end of the wound, and the entrance wound of the bullet was excised. The patient was then given one pint of blood immediately and continuous drip transfusion; he received 2,000 cubic centimetres in all over the twenty-four hours following operation. At the expiration of the twenty-four hours, glucose and saline solution were substituted for blood and continuous intravenous drip was continued till July 22, when it was discontinued. The drainage tube was removed on July 18. Penicillin was injected into the intravenous tube at the rate of 20,000 units every four hours until a total of 400,000 units had been given. Flatus and urine were passed on July 17 and at no time was distension marked. On July 18 the patient burst his incision open and a loop of intestine prolapsed into the wound. This was covered with sterile "Vaseline" gauze and continuous gastric lavage and aspiration were carried out by means of a Ryle's tube passed through the nose and connected to a syphon. This was carried out for six days, and the patient was given one ascorbic acid tablet three times a day.

On July 19 approximation of wound edges by means of through-and-through silk sutures was carried out under "Pentothal" anaesthesia. The bowels opened after a human bile enema (the patient's own gastric content) on July 22.

Convalescence was uneventful, appetite was good, the wound healed well, the bowels functioned well, though the patient complained of slight pain in the lower part of the right side of the chest. The chest was clinically and radiologically clear. On August 22 the patient was transferred by air to Adelaide for further convalescence.

Acknowledgement.

Appreciation is expressed to Surgeon Captain Carr, Director of Naval Medical Services, for his kind permission to publish this account.

CHRONIC MENINGOCOCCÆMIA.

By JOHN LANE,

Flight Lieutenant, Royal Australian Air Force.

W.V., a timber worker, aged thirty-five years, was admitted to Sydney Hospital on May 1, 1942, with a provisional diagnosis of acute rheumatism.

He had been well until seven weeks before, when he was seized with a sudden severe pain in the left hip, which forced him to retire to bed. Except for a short-lived attempt to return to work two weeks after the onset, he remained at home until admission, though not in bed all the time. For these seven weeks he suffered, with many short remissions, anorexia, pains in the limb joints and muscles and in the back and night sweats. His ankles and right knee were commonly swollen in the afternoons. From the second week onwards a very few pink maculo-papules (3.0 by 1.0 millimetres from his description) erupted on his limbs; their appearance bore no relation to pyrexial attacks. In the fourth week he had several rigors in association with the night sweats and he began to have mild frontal headaches. He had lost one and a half stone in weight at the time of admission to hospital.

Apart from an attack of probable scarlet fever at the age of twenty-eight and a mild (four days) attack of "sciatica" at the age of thirty-two, his personal and family histories were irrelevant. No significant history of recent disease in his neighbours or workmates was elicited.

Pyrexia of 102.6° F., slight pallor, apparent wasting of thighs and calves, and tenderness of the calves were the only abnormalities found on routine physical examination. No rash was present. Investigations showed the red cells to number 5,000,000 per cubic millimetre; the haemoglobin value was 14.5 grammes per centum; the white cells numbered 28,000 per cubic millimetre; of the white cells 84% were polymorphonuclear cells. The red cell sedimenta-

tion rate was 48 millimetres in one hour. The Wassermann test, the Kahn test and the gonococcal complement fixation test all failed to produce a reaction. The urine contained a faint cloud of albumin and a few pus cells and was sterile. X-ray examination of the chest revealed no abnormality.

After admission the patient had four pyrexial episodes, with recurrence of symptoms, at exactly forty-eight hour intervals. Between these attacks he felt quite well. From blood taken during the third and highest (104° F.) of these were grown Gram-negative diplococci giving the serological and fermentation reactions of meningococci. A second sample forty-eight hours later failed to yield a culture. Lumbar puncture on May 15, 1942, gave clear fluid under 200 millimetres of water pressure, with 600 cells per cubic millimetre, of which 54% were polymorphonuclear cells. A culture of meningococci was obtained, and meningococci were recovered from a throat swabbing also the following day.

On May 16, 1942, the administration of sulphanilamide in standard dosage was begun. On May 18, 1942, the white cells had fallen to 8,600 per cubic millimetre and the polymorphonuclear cells (60%) showed toxic changes. Next day the drug was suspended, thirty grammes having been given.

From May 17, 1942, until his discharge on May 28, 1942, the patient was free of all symptoms and of pyrexia. Lumbar puncture on May 23, 1942, gave clear sterile fluid with 120 cells (10% polymorphonuclear cells) per cubic millimetre. A throat swabbing failed to grow meningococci.

His white cell count had risen to 14,000 per cubic millimetre on May 28, 1942, but since he was otherwise quite well and an opportunity for suitable convalescence had presented itself, he was allowed to leave on the understanding that he would return for check-up at the end of three weeks. He failed to do this. However, he has remained well, apart from minor complaints, until the present.

It is remarkable that, though meningococci were found in the cerebro-spinal fluid, at no time did the patient show signs or symptoms suggestive of meningitis. Perhaps a terminal meningitis was about to appear when treatment was started.

Acknowledgement.

I am indebted to the Superintendent of Sydney Hospital for permission to report this case.

Reviews.

RADIATION THERAPY.

Two books have been received which from their nature demand review in company. The first, "The Physical Foundations of Radiology",¹ is a composite production by Otto Glasser, Edith H. Quimby, Lauriston S. Taylor and J. L. Weatherwax, all names which have earned a high place in American radiological literature by the sheer quality of work published in other places. The second, "The Biological Fundamentals of Radiation Therapy",² by Friedrich Ellinger, is, as its name denotes, unwittingly a complement to the first, so that the two together form a compendium of fundamental physical and biological knowledge for the practising radiotherapist.

The art of radiotherapy is based upon three sets of principles. The first of these is physical in essence and concerns the radiation or physical agent used, its character, its measurements and its control. The second set is biological and deals with the immediate and remote effects of this physical agency upon the organism, its mode of action generally and its differing effects upon various tissues and organs—really a special exercise in a small corner of biology.

It is obvious that there must be a link where the physical agency becomes the biological effect, and though complete clarity on this issue is not yet attained, our first book succinctly outlines the current views in chapters on "The Interaction of Radiation and Matter" and again in a further

¹ "Physical Foundations of Radiology", by Otto Glasser, Ph.D., Edith H. Quimby, Sc.D., Lauriston S. Taylor, Ph.D., and J. L. Weatherwax, M.A.; 1944. New York: Paul B. Hoeber, Incorporated. 7 1/2" x 5", pp. 435, with diagrams.

² "The Biologic Fundamentals of Radiation Therapy: A Text-book", by Friedrich Ellinger, M.D., with a preface by Maurice Lenz, M.D. English translation by Reuben Gross, M.D.; 1941. New York: Elsevier Publishing Company, Incorporated. Distributors: Nordeman Publishing Company, Incorporated. 9 1/2" x 6", pp. 374, with illustrations. Price: \$5.00.

chapter on the "Dependence of the Biologic Reaction on Quality and Intensity of Radiation". This is rounded out in the introduction to the second work by a discussion on the action of radiation upon the cell.

The third set of principles upon which radiation therapy is based comprises those which control the ordinary practice of clinical medicine, wherein the case is considered as a human being with a lesion rather than as a lesion in a human being. This is the practice of radiation therapy as it is used in medicine and is really the fruit of the first two sets of principles applied to the practical problem.

Our two books do not seriously touch this problem of clinical radiotherapy, but confine themselves to fundamental physics and biology. The physical book by Otto Glasser *et alii* is a delightful edition, finely produced, finely printed and a pleasure to handle. Its subject lends itself to exactness, but there is a clarity of thought in this small volume which is very welcome to the physician-radiotherapist whose primary interests are clinical and who is interested in the physics of the subject only as a means to a clinical end. In these days, the practising radiotherapist is immersed in medical detail and has no need or desire for mathematical treatises. This book supplies a definite want. It is non-mathematical, but covers the whole field of X-ray and radium physics with an adequate exposition by graph, table and text which is satisfying in the extreme. It is designed for the medical man and student rather than for the professional physicist, but even he should find it an excellent coordinating text-book for teaching purposes.

Dr. Ellinger's book deals with a less exact branch of knowledge and seems by comparison at first vague and somewhat formless. But this is only a mental illusion which vanishes if we appreciate the differences in the subject matter. Dr. Ellinger takes up the tale where the radiation strikes the cell. After a current review of the accepted and suggested effects on this primary unit he passes on to a consideration of the differential effects upon the various tissues and organs of the body. This leads to a diffuseness which is unavoidable, for there is much that is unsettled in such a field; but the author maintains an excellent balance and gives the matter as much definite form as is possible and justifiable. A just analysis shows that the achievement in this our second book is as comprehensive and as efficient as in the first.

The book is divided into five parts. The first two deal with the action of X rays and radium and of corpuscular radiation respectively. The third and fourth deal with ultra-violet radiation and ordinary light, giving a fairly complete exposition on this subject. The last section treats of general radiation biology and therapy and is of particular interest to radiotherapists.

Each of these publications can be recommended, and together they traverse the whole fundamental groundwork on which the practice of radiotherapy is based. The advantage of a separate book on each subject is that each aspect, physical and biological, is dealt with by an expert in that one particular branch. This results in a more seasoned and inspired treatment of each than can usually be obtained by the operation of one mind dealing with both branches in one book. Whoever digests both these books needs no other basic instruction in radiotherapy. The foundations on which he may build the theory and practice of clinical radiotherapy will thus be completely laid.

AS OTHERS SEE US.

MR. C. HARTLEY GRATTAN has produced an informative and valuable book about Australia.¹ In his preface he makes clear his qualifications for the task; he has visited Australia three times between 1927 and 1940 (on the second occasion with the aid of a grant from the Carnegie Corporation), has travelled widely over the country, and has made a careful study of Australia and Australian conditions generally. Incidentally, he likes us. He returned to the United States with the idea of writing two books, the first of which was to be a short introduction to the Australian Commonwealth as it is today; this is the book now under review. It should be remembered that the book was written before Japan entered the war, in 1941, and a few minor additions were made in January, 1942; thus certain statements are now out of date.

"Introducing Australia" is written for Americans, with the object of creating a better understanding of Australia

¹ "Introducing Australia", by C. Hartley Grattan; 1944. New York: The John Day Company. Sydney: Angus and Robertson Limited. 8½" x 5½", pp. 111, with illustrations. Price: 12s. 6d.

in the United States; but it should be read by every thoughtful Australian. Mr. Grattan, although he is our friend, is by no means blind to the shortcomings in us as a people and in the circumstances in which we elect to live and work. He is not afraid to be critical, and his criticisms have the value of his sincere and impartial outlook. No doubt there will be many of Mr. Grattan's comments with which Australians will disagree, owing chiefly to insuperable differences of tradition and custom. None the less, in general it will be conceded that his strictures are just, and that if we will, we may learn from them.

The chapter headings indicate the enormous amount of ground that the book covers: "As I See Australia", "As Australians See It", "A Continent", "The Making of a Commonwealth", "What Australia Lives By", "Finance and Trade", "The Standard of Living", "Governing the Country", "Cultural Life", "Oddments and Remainders", "The Tie to Britain", "The First World War", "The Second World War", "Australian Democracy", "International Position". Medical readers will see reason for regret in the fact that but little mention is made of medical and allied sciences; a short description is given of the origin and development of the Council for Scientific and Industrial Research and a word or two is written about the National Health and Medical Research Council, and there the matter appears to end. With regard to education, Mr. Grattan confines his remarks to a brief account of the school system and the universities in the chapter on "Cultural Life". However, this chapter covers a great deal of other ground—literature, art, music, the theatre *et cetera*. Attention is drawn to the "heavy export of talent of all kinds", and Mr. Grattan's remarks on this subject will be read with feeling by medical men, who are not unaware of the unfortunate fact that some of Australia's ablest scientists have felt themselves obliged to take up appointments abroad:

I see three facts which help to explain the flight of talent. One is the absence of opportunity to earn even bread and butter by the practice of many of the specialized professions, or the cultivation of non-utilitarian branches of learning, inevitable consequences of the stage of development of the country as a whole. People who get interested in such matters naturally go abroad. . . . Another point is that the nostalgia for England, reflected in the persistent use of the word "home" to mean England, sets up in sensitive minds the conviction that really to be somebody, you must become somebody at "home". A third point is related to the last. Australia has yet to evolve a centre to which the talent of the nation tends to gravitate, comparable to London in England, Paris in France, and New York in the United States.

We believe that Mr. Grattan's thoughtful study should admirably fulfil its purpose. We suggest that it should be read by Australians in the spirit in which it was written, and that they should remember that it is, after all, only an "Introduction", and therefore necessarily incomplete. Finally, we pay a tribute to the author's first-class literary style.

CLINICAL MEDICINE.

"SAVILL'S SYSTEM OF CLINICAL MEDICINE" covers the wide range of medicine, including a study of clinical methods, without being in any sense set out in tablet form. The less important subjects are in small print and the reading is easy and interesting. In addition, the more important methods of blood examinations are described.

The outstanding practical value of the work lies in its concentration on the clinical approach to disease and in the way everyday signs and symptoms are described. Each disease is preceded by a summary of signs and symptoms which, after all, are the facts known to the practitioner looking for an interpretation of his clinical findings.

There is a very useful summary of the average doses and bactericidal effects of the various sulphonamide preparations and a summary of hypnotics useful in various types of sleep disorders is given.

The present edition will help many students and practitioners over many a difficult hurdle and should more than hold its position as a book of reference and in clinical diagnosis.

¹ "Savill's System of Clinical Medicine, dealing with the Diagnosis, Prognosis and Treatment of Disease: For Students and Practitioners", edited by E. C. Warner, M.D., F.R.C.P.; Twelfth Edition; 1944. London: Edward Arnold and Company. 8½" x 5½", pp. 1196, with illustrations, some of which are in colour. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 9, 1944.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PUBLIC HEALTH DEPARTMENT OF WESTERN AUSTRALIA.

THE announcement a few months ago that Dr. C. L. Park had been appointed Commissioner of Public Health in Western Australia was received with satisfaction by practising members of the medical profession throughout the Commonwealth. Dr. Park's association with the Commonwealth Department of Health as director of the Division of Marine Hygiene, his work as chief medical officer at Australia House, London, and his experience as director of the Eastern Bureau of the League of Nations at Singapore stamped him as a man of unusual experience. The practising members of the medical profession in Western Australia therefore looked forward to his coming among them and hoped that it would result in cooperation between the Public Health Department and practising doctors and would lead to the introduction of reforms long overdue, for which they had for many years striven in vain. Soon after Dr. Park's arrival in Western Australia it became clear that if he had his way the hopes of the profession would eventually be realized. Almost immediately on his arrival in Perth he accepted a seat on the council of the Western Australian Branch of the British Medical Association and quickly became one of its most valued members. In the words of one of the senior members of the council, "his knowledge on health matters was Commonwealth-wide and his opinions and advice were recognized as sound and were eagerly sought". That in addition he was "a tiger for work" made his presence the more appreciated and hopes for the future brighter. It can thus be easily understood that the greatest concern was expressed on all sides when it was learnt at the end of October that Dr. Park had resigned his position and would soon be leaving the State. This affair is so unfortunate that publicity should be given to it and to the conditions that have brought it to pass.

Dr. Park's resignation was not known until Dr. J. G. Hislop in the Western Australian Legislative Council asked the Chief Secretary when Dr. Park's resignation would be made public, what were the reasons for the resignation, and whether Dr. Park had resigned because he thought that his efforts would be futile under the present organization. The Chief Secretary replied that the resignation had been submitted, but that no decisive action had been taken.¹ Dr. Hislop was dealing with a motion of which he had previously given notice. In this motion he drew the attention of the Government to the "urgent necessity" for the appointment of a Royal Commission, on which professional medical representation was included, to: (a) investigate the administration of the *Health Act*; (b) investigate the condition and administration of hospitals and the conditions under which nurses are trained, as well as the training they receive; (c) make recommendations for the necessary measures to be adopted during the war for the adequate hospital accommodation and treatment of all forms of sickness; (d) formulate plans for the post-war finance and provision of hospital accommodation, such plans to include the administration of hospital and nursing services. The subjects mentioned include most of the activities of the Public Health Department, and Dr. Hislop's speech in Parliament may be summed up as an expression of dissatisfaction and a charge of inefficiency against the department. In the course of his remarks he said that if there were any difficulties between Dr. Park and the lay administration they should be straightened out so that his services might be retained in Western Australia. In commenting on Dr. Park's resignation on the following day, the Honourable A. H. Panton, Minister for Public Health, said² that at his request Dr. Park had come to see him and had told him in course of conversation that "there were no complaints so far as the department was concerned, but he felt that at his age he wanted to be doing something positive in the health of the community, and Western Australia, in his opinion, was so far behind Tasmania that he felt he had not time to work up to that position, consequently he preferred to get back to Tasmania where positive work, from his point of view, was available". Mr. Panton was still hopeful that Dr. Park would reconsider his resignation.

After these happenings the Western Australian Branch of the British Medical Association decided to see what it could do in the matter. On November 9, 1944, a deputation from the Branch waited on the Minister for Public Health. The members of the deputation were Dr. H. S. Lucraft (President), Dr. F. Clark, Dr. H. Leigh Cook, Dr. J. P. Ainslie, Dr. R. D. McKellar-Hall and Dr. R. le P. Muecke. Dr. Lucraft told the Minister³ that such a speedy resignation of an officer so eminently suited to the position he occupied, as Dr. Park was, justified a request for an inquiry into the conditions of the service responsible for the resignation. Dr. Lucraft made it quite clear that in the opinion of the deputation the lay control of the department was the cause of the friction that had been present for so many years. It was a fact that for many years the Department of Public Health had been in actual practice administered by the Under-Secretary. Even the official stationery supplied for the use of the Commissioner bore the instruction: "All communications to be addressed to the Under-

¹ *The West Australian*, October 26, 1944.

² *Ibidem*, October 27, 1944.

³ *Ibidem*, November 10, 1944.

Secretary." Dr. Lucraft referred to the recommendation of the Royal Commission on Health whose report was published in 1926. In this report the following words appear: "The office of permanent head of his Department (or State Director of Health, as he should be called) should be filled by a medical practitioner highly trained in preventive medicine. No lay official should be interposed between him and the Minister." (See THE MEDICAL JOURNAL OF AUSTRALIA, January 16, 1926, page 62.) Dr. Lucraft then went on to say that the only remedy for the present state of affairs lay in a complete reorganization of the Health Department whereby the administrative control would be vested in the Commissioner of Public Health, with a secretary to advise him on matters of finance, but not to interfere between him and the Minister. In his reply the Minister said that if the Government decided on a Royal Commission, one would be appointed, but loyalty to Cabinet would not allow him to indicate his views on the subject. He admitted that he was sympathetic to the deputation's plea for professional control on professional matters; but he did not think that a Royal Commission would help matters in any way. He said *inter alia* that the Under-Secretary would be retiring from office within the next six or seven months.

On the same day on which the deputation to the Minister was reported, correspondence between Dr. Park and the Minister was released and published. Dr. Park in his letter to the Minister stated that two radical changes were necessary. The first was an increased yearly budget and the second was an alteration in the system of administration of both health and medical branches of the department. The first steps in a positive health programme, Dr. Park explained, comprised wise expenditure on maternal and infant welfare, on medical and dental supervision of the pre-school and school child and on the prevention of disease. At least an additional £10,000 *per annum* was needed to put the first two mentioned items on a good foundation. Dr. Park then dealt with tuberculosis case-finding and its cost and the remuneration of tuberculosis medical officers. He also dealt with the salaries that should be paid to other officers of the department. He finally indicated that if the Government would give a definite undertaking to do certain things in regard to financial arrangements for the department's work and for its salaries and to transfer the administrative responsibility of the department to the Commissioner on the retirement of the present Under-Secretary, he might be influenced to withdraw his resignation. The Minister in his reply defended the present policy and stated that he could give no "definite undertakings" such as those mentioned by Dr. Park. He had no alternative but to forward the resignation to the Public Service Commissioner.

From the foregoing it will be clear that Dr. Park has managed to bring to the notice of the profession and the public a great deal that should be known. The matter should be viewed if possible in a clear light that is free from the cloud of political juggling. That Dr. Park was concerned mainly with the unsatisfactory conditions on the administrative side we have no doubt whatever. Once he had handed in his resignation and was asked by the Minister for a full statement, he naturally spoke his whole mind. Had he not done so, had he, with all his experience in public health work in many spheres, gone from Western Australia without laying bare the full iniquity of lay

administration and the parsimonious, short-sighted treatment meted out by the Government to a department that should be the mainstay of the State in its future progress, he would have failed in his duty and would have earned the censure of his brother practitioners and of the community. In fact, the very politicians who at present seem to attribute mercenary motives to him, would have been the first to blame him, if it had suited them to do so, had he left the State without giving to the people the benefit of his experience. One can almost hear the righteous indignation that would well up in such a case. That the medical profession in Western Australia has in the past urged reforms on the Government has been made clear by Dr. Lucraft in one of his letters to *The West Australian*. Ever since 1932 it has been urging the Government to appoint a hospitals commission. The Government has recently invited the Western Australian Branch Council to appoint a medical practitioner to a departmental committee which has been collecting data regarding hospital requirements for the purpose of formulating future hospital policy. The Branch Council has reminded the Minister of its past efforts to secure the appointment of a hospitals commission and has urged him to accept two representatives on the committee—one to be a man versed in hospital organization and the other to be a leading member of the practising branch of the profession. The Minister, we learn, has stated that he is willing to accept two medical appointees. At the same time the request for a representative is belated and will not be looked on by the profession as in any way an offset to its demand for a Royal Commission. It is time that the people of Western Australia arose in a body and demanded a full and complete investigation into the activities and the administration of its Department of Public Health. Health is man's greatest asset, and the people of Western Australia should see to it that conditions are most favourably set for its attainment.

Current Comment.

PROBLEMS IN AMOEBIASIS.

AMOEBIASIS is now known to be a cosmopolitan disease, though its incidence and severity are greater in tropical areas. It is also now well known that it may be the underlying cause of varying clinical states such as general ill health or vague abdominal discomforts, quite apart from the more dramatic features of dysentery or hepatitis. But there are many riddles not yet solved, and some of these are stated and discussed in Ernest C. Faust's Alvarenga Prize Lecture.¹ One of these questions concerns the possibility that amoebiasis may be caused in man by infection from other mammals. Kittens are known to be readily susceptible to amoebic infection, often of a fulminating type, but there is no evidence that the disease occurs in cats naturally. Dogs sometimes suffer from isolated infections or small epidemics may occur among them, but Faust from his own extensive observations and those of others does not think dogs are a source of danger in this regard to human beings. Other mammals appear to be merely incidental hosts. It might be thought that the mode of infection was clearly understood, but the author points out gaps in our knowledge. Water, food, flies and direct contact are the methods of spread universally accepted;

¹ *Transactions and Studies of the College of Physicians of Philadelphia*, December, 1943.

evidence is chiefly epidemiological, for direct proof is hard to obtain. No one doubts that water can carry the *Entamoeba histolytica*, as experiences in Manila and Panama show, and especially such a localized outbreak as the famous one in Chicago in 1933. Yet cysts were not actually isolated from the indubitably contaminated water in this last instance. Similarly the evidence about food is indirect. Some American surveys of food handlers have shown a higher incidence in this group, and treatment of infected food handlers has on occasion lowered the incidence rate in the general population served by them, but Faust quotes a fairly recent survey in the Navy in which they did not seem to increase the hazard to others. With regard to flies there is a little direct evidence of carriage of infection, and in any case there can be no doubt as to the correct view to take. Direct contact is also possible, and the studies of Ivanhoe are quoted in which cysts have been identified in clothing, toys and the water of a wading pool in an infant asylum. All these points make it hard to assess the risk of cyst carriers in a community. It has been argued that the cysts found in routine surveys of stools, even if without doubt those of the genuine *entamoeba*, may not be virulent. There is, however, experimental and other evidence that they are not all avirulent. Therefore there are two questions of importance: how are the cysts disseminated to others, and what factors determine their capacity to cause clinical disease?

With regard to the latter problem, Faust first traverses the known facts related to the pathology of invasion. He points out that a little colony of *amoeba* may be established in the mucosa of the colon within twenty-four hours of initial contact. Thereafter it invades the submucosa and spreads laterally to produce the familiar bottle-neck lesion. From this site extension to blood channels occurs, which may in turn be followed by more extensive local lesions both ischemic and necrotic in causation, or by embolic spread to the liver. It is known that simple invasion may be multiple and not be associated with bacterial infections, but on the other hand local inflammations due to bacteria, such as those of bacillary dysentery, may be responsible for determining the clinical onset of disease. Thus an outbreak of mild bacillary dysentery in a group with latent *amoebic* infection might make manifest several new cases of *amoebiasis*. Faust believes that in mildly endemic areas ingested cysts may actually exist in the ileum, but never manage to establish themselves in the large bowel. Heavy cyst infection is, of course, a definite hazard, and if this occurs in a significant number of people simultaneously the stage is set for an epidemic.

The question of nutrition must also be of importance. Faust states that the most important single intrinsic factor which determines whether an *amoebic* infection is acute, chronic or relatively asymptomatic is the degree of resistance based on the nutrition of the animal. He points out that research has shown that carbohydrates appear to favour the multiplication of the *amoeba* and animal proteins to lower it. Further the anaemia observed in the course of the infection in animals was controlled by fresh liver or the ordinary liver or stomach extracts and concentrates, but whole liver or freshly expressed juice had a favourable effect on the *amoebiasis per se*. Even more interesting is the finding that fresh liver juice effectively controlled the infection in the colon of laboratory animals when applied locally by a retention enema. There would seem to be a valuable field for exploration here, especially when we reflect that it is believed that most of the *amoebae* reaching the liver as emboli fail to colonize. Doubtless there are many considerations related to races, age, exposure, nutrition *et cetera* to be taken into account, and in addition there may be some questions of specific tissue resistance.

This paper is too highly compressed to be summarized easily, but in conclusion it may be noted that the author's experience with diagnosis shows that a single faecal film has revealed only 20% to 25% of positive findings in cases ultimately proved to be *amoebic*, whereas a method including use of a dried wet film, a stained film and flotation film demonstrated the *amoebae* in 85% to 90% of cases. This author is highly qualified to write on this subject, and though he admits that the material in his

paper is not all intended for immediate practical application, he has provided some stimulus to those interested in this condition.

PERSONALITY CHANGES IN THE DIABETIC CHILD.

ONE of the prime functions of a doctor is to help the sufferer from a chronic malady to integrate his life to his complaint. This is not always successfully done, and it is reasonable for the doctor to blame himself somewhat if the necessary restrictions imposed on the patient leave him in spiritual bondage. Even greater than the problem of the adult subject of chronic disease is that of the child similarly afflicted. All who have dealt with diabetic children have probably been impressed with the unspoiled cheerfulness that blesses most of them. Winifred C. Loughlin and Herman O. Mosenthal have made a study of the personalities of children with diabetes based largely on observations made during a summer camp conducted for diabetic children by the New York Diabetes Association.¹ The children ranged in age from six to eighteen years and numbered 114. About three-fifths of them showed no departure from norms of child personality. One may ask what this norm is. The authors' definition is "possession of a sense of security, acceptance by a group, interests in the activities of a group, a healthy attitude toward bodily functions without pre-occupation by them, absence of the urgent need for indulgence at a given moment and ability to endure a moderate amount of deprivation". This seems a very comprehensive standard, based on data related to behaviour, which seems a rational way to judge children. The average duration of the diabetic state in these boys and girls was 4.3 years, and no substantial difference in duration was noted in the normal and abnormal. The children judged abnormal were not marked out by any special feature in their medical history or condition, except that ketosis was twice as common among the abnormal children as among those well adjusted emotionally, and the diabetes occurred rather earlier in life. The authors wisely comment that the earlier onset possibly is more disturbing to personality, whereas the greater tendency to ketosis may in part be due to greater instability. The abnormalities noted among these children ranged from true neurotic states to simple disturbances of personality. It is not surprising that cases have occurred in which the mere antidiabetic propaganda of these camps has caused greater preoccupation with the disease. Nor is it at all surprising that such experiences have led the organizers of the camps to reduce the number of examinations of urine from four per day to two. The varieties of abnormal personality encountered are listed as the aggressive, the retiring, the devil-may-care, the immature and the escapist. In fact, one may say, we have the adult here *in petto*. Naturally the devil-may-care and escapist groups are dangerous to their own well-being and may have a bad influence on others. Of course, there is an obvious weak point in an analysis like this, that is, its lack of statistical comparison with normal groups. But that really does not matter much. What matters is that it should be realized that the psychologically unstable child with diabetes may present difficult problems in handling, whether these arise out of his own natural make-up, his diabetic state or his environment. A good point is made by Loughlin and Mosenthal when they state that the individual handling of each child, so necessary in this disease, is made easier now that insulin has relieved us of the need or danger of making the child too conscious of food. We must applaud this statement: "The day of weighing of food is over and measuring resembles the normal every-day way of dispensing food even in the home." There is no reason why the diabetic should not pursue an average type of life, or why he should not be well adjusted to it, and thoughtful consideration of the little patients who face adult life with this handicap should greatly help them in the problems that lie ahead.

¹ American Journal of Diseases of Children, July, 1944.

Abstracts from Medical Literature.

THERAPEUTICS.

Operational Fatigue.

D. W. HASTINGS, B. C. GLUECK AND D. G. WRIGHT (*War Medicine*, June, 1944) describe the use of "Sodium Amytal" narcosis in the treatment of operational fatigue developed in a number of airmen engaged in aerial bombardment. It resulted from repeated exposure to the harrowing experiences, tension, fatigue and lack of sleep during the course of operational tour. It developed in fundamentally stable men, and did not imply lack of courage nor desire to be removed from combat status. Some 12 to 18 heavy bombardment missions, flown approximately bi-weekly, each lasting about seven hours, with about four hours spent at altitudes above 25,000 feet (7,500 metres), frequently gave rise to operational fatigue. The patients were usually pale and heavy-eyed; they appeared depressed. Irritability over trifles was always present; lack of sleep and nightmares were usual. Anxiety was either general or related to a particular aspect of combat flying, such as "flak" or fire on board or enemy fighters. Tremor, anorexia, nausea and vomiting, and a loss of weight (average twelve pounds) occurred in 50%. Tachycardia, palpitation, sweating, giddiness and pale extremities were frequent. With these symptoms a man might be unfit for three months. The lack of sleep prevented recovery. A method of treatment was evolved in which 0.2 to 0.4 gramme of "Sodium Amytal" was given every night for a few nights, and a watch was kept for signs of sensitivity to the drug. If no such signs developed, a cathartic was given, and twenty-four hours later 0.4 to 0.6 gramme of "Sodium Amytal" was given and the dose regulated and continued so as to ensure twenty hours of sound sleep in every twenty-four. This narcosis was continued for thirty-six up to ninety-six hours according to the severity of the symptoms. The average duration was seventy-two hours. In several cases a second forty-eight hours of treatment was given when symptoms persisted. Blood pressure was taken every half hour; the systolic pressure fell to 80 or 90 millimetres of mercury during narcosis. The pulse rate was 60 to 70 and respirations numbered 18 to 20 per minute. A systolic blood pressure below 80 millimetres of mercury, and dilatation of the pupils with reversal of the light reflex indicated danger. Oxygen 100% given by mask for ten minutes restored the patient. Picrotoxin was available, but was never used. Very careful nursing was necessary to ensure adequate food intake, the patient being turned every half hour to prevent stasis in the extremities, atelectasis or hypostatic pneumonia. If the bladder was not emptied after twelve hours, catheterization was necessary. For forty-eight hours after narcosis, the patient had to be watched carefully, as he might be ataxic, have difficulty in focusing his eyes, might fall or burn himself. To ensure sleep 0.4 or 0.6 gramme of "Sodium Amytal" was usually given each night, but was stopped completely six or seven days

after cessation of narcosis. Graduated exercise followed, and return to full normal activity in a week. Throughout the course encouragement and psychotherapy were necessary. An additional week of rest and recuperation before return to duty was often helpful. Sixty-nine patients were treated: 70% resumed combat duty, of whom ten patients completed their operational tour; 25% returned to ground duties.

Thiouracil and its Effects upon Hyperthyroidism.

J. K. MCGREGOR (*The Canadian Medical Association Journal*, July, 1944) reports his observations upon the results of thiouracil treatment in twenty cases of hyperthyroidism. He states that thiouracil lowers the basal metabolic rate faster than does iodine in the form of Lugol's solution, that a feeling of improvement occurs within a few days after its administration, that some patients are as well within six weeks as if they had been subjected to thyroidectomy, that the drug seems ideal for post-operative recurrent hyperthyroidism, that it appears to be the ideal therapeutic agent for the patient who is not a good operative risk, such as the hypertensive and the psychotic, and that previous iodine treatment retards the effect of the drug. The author has not observed any of the reported ill effects from the drug, and the lowest white blood cell count noted was 4,600 per cubic millimetre. He suggests that a certain group of patients with hyperthyroidism may not have to be subjected to thyroidectomy if afforded treatment by the drug.

ROBERT H. WILLIAMS AND HOWARD M. CLUTE (*The New England Journal of Medicine*, June 1, 1944) have used thiouracil in the treatment of 72 thyrotoxic patients, including patients with classic Graves' disease, toxic nodular goitre, and toxic adenoma. The duration of the illness varied from three weeks to twenty-two years. Subtotal thyroidectomies had previously been performed in thirteen of the cases. Thirteen patients had taken potassium iodide for several weeks, discontinuing it within about a month before thiouracil therapy was begun. Most of the patients were not in hospital for more than one day, and performed their usual duties. The period of observation has been between two and six months. Thiouracil lowered the basal metabolic rate to a normal range and maintained it at this level so long as treatment was continued. There was a clinical remission of the disease with disappearance of tachycardia, hyperidrosis, nervousness, diarrhoea, weight loss, and other toxic manifestations. The ideal dosage is the smallest amount necessary to obtain a remission of the disease, but this quantity varies with different patients. The authors used 0.2 gramme three times daily for the first two weeks, followed by 0.2 gramme twice daily until the metabolism was normal, and then 0.1 gramme twice daily until the basal rate had been maintained well within normal limits for several weeks, when the dosage can be reduced to 0.1 gramme daily. Smaller doses were used in a number of cases. Patients who had had iodide previously responded more slowly to thiouracil than did those who had not had any. Furthermore, in some cases there was actually an exacerbation of the disease if thiouracil was suddenly exchanged

for iodide. A period of preparation of four or five weeks with thiouracil before operation has proved satisfactory. Complications of thiouracil therapy consisted of agranulocytosis (one case), morbilliform rash, urticaria, allergic arthritis, oedema of the legs, vomiting and enlargement of the submaxillary salivary glands.

Thiouraea.

E. D. GOLDSMITH, A. S. GORDON, G. FINKELSTEIN AND H. A. CHARIPPER (*The Journal of the American Medical Association*, July 22, 1944) describe experiments in therapy to prevent granulocytopenia induced by thiouraea. Thiouraea and thiouracil are effective in treatment of hyperthyroidism owing to their interference with the synthesis of normal thyroid hormone. However, these drugs cause agranulocytosis in some cases. The experiment reported consisted in feeding 0.5% thiouraea to rats for 58 days; another series of rats were given 5% solubilized liver in addition to thiouraea, and a third series of rats, untreated, served as controls. Blood counts showed that the total white cell count significantly decreased after forty days on thiouraea therapy, and a marked neutrophilic granulocytopenia developed which became progressively more severe with treatment. Administration of liver almost completely prevented the development of neutropenia. It has been shown that the active factor in liver is folic acid. It is suggested that the neutropenia due to sulphonamides or thiouraea could be prevented by giving liver or folic acid.

Digitalis and Thrombosis.

G. DE TAKATS, R. A. TRUMP AND N. C. GILBERT (*The Journal of the American Medical Association*, July 22, 1944) discuss the effect of digitalis on the clotting mechanism. The cause of emboli in auricular fibrillation apart from the effects of severe stasis in the auricle has often been discussed. It has been said that digitalis shortens coagulation time both *in vitro* and *in vivo*. The onset of embolic phenomena following increase of digitalis dosage has been observed. The authors record four such cases. They also record observations which indicate that patients showed increased resistance to heparin while under digitalis therapy, and that this resistance decreased rapidly as soon as digitalis was stopped. Experiments on dogs showed that coagulation of the blood was retarded after intravenous administration of heparin, and that it was increased after digitalis. The suggestion is that digitalis favours thrombosis and so gives rise to emboli in auricular fibrillation.

NEUROLOGY AND PSYCHIATRY.

Cystic Hydrops of the Pineal Gland.

Cystic developments within the pineal body are, according to Jesse L. Carr (*The Journal of Nervous and Mental Disease*, May, 1944), generally divided into three types: (i) small single or multiple cavities which do not cause enlargement of the gland, (ii) cysts associated with tumours, and (iii) cysts unassociated with tumours which cause enlargement of the gland, and pressure on contiguous territory. The author presents six cases of the last-

mentioned class together with a discussion on pathogenesis. Post-mortem findings are also given. In none of the cases reported either in earlier or present communications was the diagnosis made prior to death, although in one case an intracranial lesion was suspected. Two of the cases under review were associated with mental disorders; two patients reached the age of sixty years and died suddenly while in apparently good health. Various neurological symptoms displayed in cases of cystic hydrops may be explained by pressure on neighbouring structures; but there is no physical basis to explain the functional changes other than cortical oedema.

Clinical Allergy in the Nervous System.

CLINICAL allergy in the nervous system is discussed by Albert H. Rowe (*The Journal of Nervous and Mental Disease*, May, 1944), who states that serum allergy may cause cerebral symptoms, and especially transient paralysis in the peripheral nerves. He believes that macroscopic and histological lesions produced in experimentally sensitized animals indicate how the nervous system may be affected allergically. Allergy to food may cause transient stupor or coma, migraine and recurrent or chronic headache. The question of allergic epilepsy is considered. Ménière's syndrome and dizziness may be due to allergy in the inner ear and its nerves. Allergic toxæmia causing fatigue, stupor and nervousness, and irritability, nervousness and sleep disturbances in children are believed to be due to chronic allergy, especially to certain food. The diagnosis and control of clinical allergy of the nervous system are discussed.

The Bruns Syndrome.

BERNARD J. ALPERS AND H. E. YASKIN (*The Journal of Nervous and Mental Disease*, August, 1944) claim that the symptom first described by Bruns in 1902 now represents a definite syndrome. In their opinion, this is characterized by the following three features: (a) vertigo, vomiting, headache, and visual disturbance with change of posture of the head; (b) freedom from symptoms between the episodes; (c) posturing of the head. These authors present and discuss five cases illustrating the Bruns syndrome. They claim that it is most often described in association with cysticercus of the fourth ventricles; but it is also found in tumours of the mid-line of the cerebellum and with third ventricle and lateral ventricle tumours. Though the generally accepted explanation of the symptoms has been the blockage of the ventricular system on change of head posture, the authors' pathological studies suggests a possible disturbance in the vestibular mechanism in the brain stem.

Return of Virility after Prefrontal Leucotomy.

R. E. HEMPHILL (*The Lancet*, September 9, 1944) reports the case of a man who had suffered from a long-standing obsessional neurosis with emaciation, impotence and hypogonadism and on whom the operation of prefrontal leucotomy was performed. Within four months, following the operation, this patient became obese, with gonads of normal size and normal

sexual behaviour. The patient, a man of thirty-three, had been impotent since the age of twenty-eight, and at the time of operation was confined to a mental hospital suffering from pituitary insufficiency leading to atrophy of the testes (confirmed by biopsy) and emaciation. The writer believes that in severing the white fibres passing dorsally from the frontal cortex, the hypothalamus or pituitary was released from inhibitory influences.

Bird Language in Schizophrenia.

AUDITORY hallucinations, as L. Kerschbaumer contends (*The Psychoanalytic Review*, April, 1944), are an accepted symptom of schizophrenia; but they are generally human "voices". He quotes two cases in which the patients maintained that they understood bird language and that birds spoke to them and directed their conduct. The first case was that of a male of thirty-nine years whose history showed him to have been a petty criminal and waster. Among other psychotic symptoms he claimed to understand bird language. He would divulge no details, but would sit listening and grinning to himself. The second case was that of a female, aged twenty-five years. She also was of the vagrant type, with a history of sexual promiscuity and alcoholism. Her intelligence quotient was 73. She maintained that the birds told her what to do, and she had to do it. When her psychotic condition improved, under the influence of "Metrazol" injections, and she was asked to explain the bird language, she became embarrassed and said "twit twit".

Fatal Circulatory Failure Caused by Electric Shock Therapy.

REPORTING three recent cases of fatal circulatory failure following electroshock therapy, Walter W. Jetter (*Archives of Neurology and Psychiatry*, June, 1944) draws attention to this danger and discusses the probable manner in which death is brought about. He believes that there are at least two mechanisms involved in the production of fatal circulatory failure. In the first place, he presumes that the violence of the shock itself may cause cardiac dilatation, especially in patients suffering from cardio-vascular degeneration. In the second place he considers that a fatal termination to shock may ensue in patients displaying no gross signs of cardiac disease. In such cases he suggests that death may result from the electrical stimulation of hyperexcitable central cardio-regulatory or vasomotor centres. The existence of such a centre, he admits, must be regarded as hypothetical rather than proved. Of the patients reported in this communication two suffered from advanced arteriosclerotic heart disease and the other from acute myocarditis.

Sweat Secretion in Man.

CARL FELIX LIST AND ALOYSIO DE MATTOS (*Archives of Neurology and Psychiatry*, June, 1944) report three cases in which spinal reflex sweating appeared as a symptom of a transverse lesion of the spinal cord. In the first case, there was total absence of thermoregulatory sweating, but spinal sweating which was present in the upper half of the body was later abolished by sympathectomy. In this patient the spinal sweating response

could be elicited by distension of the bladder, by external pressure on the suprapubic region and by the patient's assuming a sitting posture. In the second case thermoregulatory sweating of the head and neck only was present, with spinal reflex sweating of the trunk and inner part of the arms. The lesion of the cord was at the level of the third and fourth dorsal segments. In the third case, where a transverse lesion existed at the level of the eleventh thoracic segment, thermoregulatory sweating was preserved over the entire anterior surface of the body, with spinal sweating of the lower extremities and the abdomen. The authors maintain that spinal sweating is a release phenomenon and a component of the spinal mass reflex; hence it may be induced by any afferent stimulus acting on the distal portion of the spinal cord. Relief from excessive spinal sweating may be obtained by sympathectomy.

Schizophrenia in a Four Year Old Boy.

Dementia præcoxissima is a rare illness, as H. Robert Blank, Olive Cushing Smith and Hilde Bruche (*The American Journal of Psychiatry*, May, 1944) point out in presenting such a case occurring in a male child of four and a half years. The parents were refugees; the father an extremely neurotic, frustrated and even paranoid individual and the mother a schizoid, almost as sick as her own child. The child was from birth subject to severe emotional disturbance and social limitations in a home atmosphere full of tensions and threats. The parents were deeply involved in their own emotional conflicts. The father's attitude to the child was one of rejection; the mother's attitude was that of marked over-protection; and the authors believed that the child's symptoms might be regarded as defensive stratagems giving him protection from a threatening world. A detailed clinical description of the child's behaviour is given during the nine months he spent in the child study centre. Treatment of the mother had also to be undertaken; and when she achieved a better adjustment, she had the child at home with her. The prognosis in these cases is uniformly poor; and the authors are of the opinion that little can be expected other than a fair adjustment to simple living conditions with ultimate mental deterioration.

The Fear of False Teeth.

H. S. DARLINGTON (*The Psychoanalytical Review*, April, 1944) goes deeply into the anthropological and psychoanalytical aspect of teeth and quotes from the case of a man whose teeth were unconsciously linked with a repressed recollection of some early attempt at incest with his sister. False teeth in this man's case were rejected because in his unconscious mind the wearing of them would deprive him of going to heaven. The writer quotes extensively from folk-lore and anthropology to show the symbolic significance of teeth, and how, together with hair and nails, they are related to the hope of resurrection from the dead. The patient in question unconsciously regarded his lost teeth as the cast-off, illegitimate offspring of his incest relationship, for he found (without knowing the reason) that he could not eat food offered by his sister while retaining his false teeth.

British Medical Association News.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed civil practice as from the dates mentioned:

Dr. S. V. Marshall, "Harley", 143, Macquarie Street, Sydney (July 1, 1944).

Dr. John Gray, 2, Collins Street, Melbourne (November 20, 1944).

Dr. L. R. Jury, Big Bell, Western Australia (November 9, 1944).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on November 17, 1943, at the Children's Hospital, Carlton, Melbourne, Dr. W. FORSTER, the President, in the chair. Part of this report was published in the issue of November 25, 1944.

Slipped Epiphysis.

DR. WILFRED FORSTER showed a female child, aged thirteen years and five months. Two months earlier she had fallen over in a gutter, and two small girls had fallen on top of her. The child experienced no discomfort immediately after the fall. Two weeks later, when putting on her shoes, she found that she was unable to lift her left leg, and about this time she complained of pain in her left hip, which was aggravated when she jarred her leg. She then developed a limp which gradually became worse, and the left knee became stiff because of her limp. Her general health had been good, though her appetite had always been poor and her weight variable. On examination, she was a thin child, who walked with a pronounced limp; but general inspection revealed no abnormality. Pronounced tenderness was elicited over the left hip joint, with much limitation of movement in all directions. There was limitation of flexion at the right hip joint because of pain in the left. The left leg appeared to be shorter than the right, but was not actually so. One inch of wasting of the thigh was found two inches above the patella, and half an inch of wasting was found five inches above the patella.

The first X-ray picture was taken in the antero-posterior plane only, and the radiologist reported no bony changes. However, the X-ray examination was repeated two days later and included a lateral view of both hips. In the latter view a slipped epiphysis could easily be visualized. Dr. Forster emphasized that the diagnosis would have been missed if reliance had been placed on the straight X-ray picture only.

DR. KEITH HALLAM said that cases of slipped epiphyses were interesting and rare. He recalled with pleasure the conversations he used to have with Dr. Charles Osborne, who taught him a great deal about lesions of the hip joint. They had discussed in particular an article by Milch, which had appeared about six years earlier. In this work, the mechanism of slipped epiphysis had been described in some detail. Milch described a "double epiphyseal line". Although it was not striking, there was a suggestion of this line in Dr. Forster's case. Dr. Hallam pointed this out to those present on the X-ray films taken, and demonstrated Milch's "double line" by means of a diagram on the blackboard.

DR. H. DOUGLAS STEPHENS said that George Perkins, of Saint Thomas's Hospital, London, held that the epiphysis on the affected side was blurred and thickened. Moreover, an overhanging or lip developed, the presence of which was highly suggestive of the disorder. In Dr. Forster's case there was a suspicion of overhanging on the right side as well as on the left. Dr. Stephens said that a second epiphysis could slip whilst the child was in bed. If only one side was affected, he advised avoidance of weight-bearing for twelve months. If the lesion occurred on both sides, the child had to be kept in bed, as weight-bearing could not be achieved.

Dr. Forster, in reply, said that he had not had much experience in this type of case. He remembered another child, in whose case Dr. Wait had made the diagnosis. The patient was kept in bed in a position of abduction for nine months, and then was gradually encouraged to walk with a hanging leg for three months. A series of X-ray films taken subsequently showed that the epiphysis had become established in the correct position, and after eighteen months the child was able to walk without disability. Dr. Forster said that if only short periods of treatment were given, the hip would slip further and the disability would be greater. Not much manipulation was required; but it was necessary to put the limb in a position of wide abduction.

Pyloric Obstruction.

DR. ROBERT SOUTHEY showed three children, in whom symptoms of pyloric obstruction had appeared. The first was a male child with a history of vomiting every day since birth; he vomited after at least two feedings per day, and sometimes more frequently. The birth weight was eight pounds two and a half ounces. The baby was breast fed for the first three weeks of life and then was given a patent preparation for the next three weeks. The vomiting was not projectile. The baby was constipated. At six weeks he weighed seven pounds six ounces. No peristaltic waves were visible, nor could a tumour be felt in the abdomen. Fluoroscopic examination after a "Basilac" feeding revealed no residue in the stomach after four hours. Ten days later, however, peristaltic waves were visible after feeding, and a pyloric tumour was felt. A further fluoroscopic examination four hours after an opaque meal showed that half the feeding was still in the stomach. Ramstedt's operation was proceeded with, and a small vascular pyloric tumour was found and divided. The baby's weight at this time was seven pounds fifteen ounces. Vomiting persisted after the operation, but with diminished frequency, and the baby gained in weight. One month after the operation, at the age of three months, the baby was discharged from hospital, weighing nine pounds eight ounces.

He was readmitted to hospital two months later, as the vomiting had not abated. Two weeks previously he had vomited two mouthfuls of bright blood, and since then the vomitus had contained brown slime. On about four days in the week the vomiting was projectile in nature. The bowels opened regularly, though the stools were hard. Fears were entertained of mechanical obstruction. At the age of eight months some vomiting was still present and the bowels became obstinately constipated. The baby then weighed twelve pounds six ounces. Examination at the time revealed great distension of the abdomen. Scybalous fecal masses were palpable in the right iliac fossa, in the ascending colon and in the left iliac fossa. Hard fecal masses were felt on rectal examination. The baby was given regular enemata and bowel washouts. Occult blood was present in the feces. Examination after a barium clyisma revealed dilatation of the whole of the colon. Examination after a barium meal showed that some barium remained in the stomach after twelve hours. After thirty-six hours all the barium was in the rectum. The diagnosis was considered to be neuromuscular incoordination. Treatment consisted in the administration of five cubic centimetres of "Eumydrine" (1/10,000 solution) before each feeding. A rhubarb and soda mixture was also administered, and the establishment of bowel control was attempted by the use of paraffin oil and daily enemata. On this régime the baby steadily gained in weight. At the age of thirteen months he weighed nineteen pounds. Examination after a barium meal at this time revealed no delay in oesophageal emptying. After six hours there was no residue in the stomach. No abnormality could be demonstrated in the stomach and duodenum. At the age of nineteen months occasional vomiting was reported. Examination after a barium meal at this time revealed no abnormality in the alimentary tract. At two years, the child weighed twenty-eight pounds three ounces, and he was comparatively well, though he showed little appetite for solid food.

The second baby shown by Dr. Southby was a male, aged thirteen months. At birth, the baby weighed eight pounds. He was breast fed at intervals of three hours. Vomiting began at the age of three weeks and occurred every day. During the first three months of life, the bowels opened frequently five or six times a day, and the stools were relaxed. At the age of four months the baby had a pale, wasted appearance. The stomach appeared to be much dilated, and peristaltic waves were visible travelling across the epigastrium. The breast milk supply at this time was obviously inadequate, and it was supplemented with a

suitable patent preparation. The child's weight was ten pounds five ounces and the haemoglobin value was 116%. The erythrocytes numbered 6,900,000 and the leucocytes 9,800 per cubic millimetre. No abnormality was detected in a blood film. Examination of the gastric contents revealed ample supplies of hydrochloric acid. Examination after a barium meal showed most of the barium to be still in the stomach after four hours; another such examination revealed the outline of a greatly distended stomach. Even after twenty-four hours, there was still some barium in the stomach. The appearances in the films suggested pyloric obstruction with resulting gastric dilatation. The baby was treated with tincture of belladonna, two drops in one teaspoonful of water, ten minutes before each feeding. On this therapy the vomiting was reduced to a minimum and the bowel actions became less frequent. One month later, three drops of tincture of belladonna were ordered before each feeding. The baby proceeded to gain in weight in spite of occasional vomiting. At eleven months a barium meal examination still revealed great distension of the stomach with partial pyloric obstruction. At the age of thirteen months, the baby was quite well and free from vomiting, and weighed 24 pounds 14 ounces.

Dr. Southby's third patient was a female child, aged eighteen months. She was the second child of normal parents. Delivery was normal, and the baby weighed eight pounds at birth. She was breast fed for four months, and then her feedings were supplemented with a patent preparation. In the early months of life the baby was noticed sucking cotton or threads. At twelve months she began to pull out the hair of her scalp and eyebrows and to eat it. At this time she was fretful and bad-tempered. No vomiting occurred. Hair appeared in the stools, which were occasionally specked with blood. The development of a hair ball in the stomach was suspected. At fourteen months a barium meal was given, and examination revealed that most of the barium was still in the stomach after six hours. The stomach was dilated and atonic owing to pyloric obstruction. Treatment consisted in the administration of phenobarbitone, bromide and "Syrup Minalex". The habit of eating hair gradually disappeared. At the age of eighteen months the child was well, and weighed 25 pounds nine ounces.

DR. H. D. STEPHENS said that Dr. Southby's first case was one of pyloric stenosis, in which a neuro-muscular condition became superimposed subsequently. The second case was a triumph for the use of belladonna. The third case might reasonably have been one of hair ball in the stomach. Dr. Stephens said that he remembered a child who ate his clothes as well as his hair. The child was subsequently operated on, and a perfect cast of the stomach was found. In the intestine there were two other balls, which were worked down to the large intestine and finally removed by enema.

DR. W. FORSTER said that one should not refrain from terming the second case one of pyloric stenosis because recovery occurred without surgical interference. Indeed, the contrary was more probable. With regard to the third case, a decision concerning the presence of a hair ball might be reached by examination under anaesthesia. This method of examination in doubtful abdominal conditions had a great deal to commend it.

DR. HENRY SINN said that in Melbourne the chosen method of treatment for pyloric stenosis was operative. However, in the type of case in which the onset was late, or in which treatment was required in the third month (as in Dr. Southby's second case), it was possible to "coast" the patient along with antispasmodics. The Scandinavian school had demonstrated beyond doubt the efficacy of these drugs, even in true and early pyloric stenosis. The great disadvantage was the long stay in hospital required, with the associated danger of cross-infection. Surgically treated patients could be discharged from hospital in ten days, and ideally isolated at home during the convalescent period. With medical treatment, an average stay in hospital of seventy-two days was required. Medical treatment had received a great uplift when "Eumydrine" was introduced; this was an atropine preparation of high potency and low toxicity. It was given in solution, diluted to one in ten thousand, and the usual dose was 0.5 to 1.0 drachm fifteen minutes before feeding. Unfortunately the supplies of the drug in Australia had been exhausted, and it was not now available. Other less reliable preparations were atropine sulphate solution (one in 1,000) or tincture of belladonna, one minim in a teaspoonful of boiled water, fifteen minutes before the feeding, the dosage being gradually increased by one minim at a time until the desired effect was produced or until toxic symptoms supervened. These symptoms were dryness of the mouth,

dilatation of the pupils and sometimes a rash, erythematous in type. In his second case, Dr. Southby had demonstrated how cure could be obtained by the judicious use of antispasmodics (in this instance belladonna drops) even in severe cases. It might be observed that in this case the child was first brought for diagnosis at the age of four months, though the vomiting had begun at three weeks.

Dr. Southby, in reply, said that he thought the group made an interesting triad. He believed that the palpation of a tumour mass was the point which decided medical or surgical treatment. Dr. Southby said that it was amazing how rarely complications followed pyloric stenosis. He recalled a girl, aged three years, who had been successfully operated on for pyloric stenosis as a baby. Vomiting and constipation developed, and she presented the picture of intestinal obstruction. She was admitted to hospital with a provisional diagnosis of intestinal obstruction from adhesions; but she eventually proved to have a cerebellar tumour.

Correspondence.

CANCER OF THE CERVIX UTERI.

SIR: My reason for writing a previous letter to you was to give constructive criticism of the paper by Dr. Schlink and Dr. Chapman on "Cancer of the Cervix Uteri", and to point out that some of their conclusions based on their presented statistical tables were hardly permissible. It is unfortunate that they should infer in their letter in your issue of November 11 that radiotherapists have been guilty of "manipulation of facts published by their opponents". I would prefer that my gynaecological colleagues were earnest collaborators with the radiotherapists in a well-organized cancer unit.

Dr. Schlink and Dr. Chapman have quoted from the excellent chapter on cancer of the uterus (I use their own words) in "Recent Advances in Obstetrics and Gynaecology" by Bourne and Williams. It is a pity that they did not quote for the benefit of your readers the conclusion reached by these writers after a thorough discussion of numerous statistics. Their words are: "It hardly seems possible to avoid coming to the conclusion that, generally speaking, radium can do everything that surgery can in the attempt to cure cancer of the cervix. . . . The figures of the end results must be allowed to speak for themselves. On the precepts of the best good for the greatest number, radium seems to be the method of choice."

Dr. Schlink and Dr. Chapman in their letter refer to the Christie Hospital and Holt Radium Institute, Manchester, and to the fact that this centre "could only present 205 five year survival cases in their report compiled in 1939". They do not point out that these cases were seen in two years only, that is, 1932 and 1933. These years were the first that the Christie Hospital and Holt Radium Institute were organized as one unit. The number of cases of this disease presenting at this clinic has been for some years now 200 or over *per annum*. When radiotherapists abroad see numbers of cases of this order, year in and year out, it does not seem logical that Dr. Schlink and Dr. Chapman should infer that radiotherapists have insufficient clinical knowledge of this disease.

My comment on absolute statistics at Royal Prince Alfred Hospital has I think been misunderstood. I am quite conversant with the meaning of the term. I was referring to the fact that under our present system, cases of this disease coming to a large general hospital are segregated to various clinics. Some of the advanced cases not considered suitable to be referred to a predominantly surgical clinic are referred direct to the deep X-ray department. This has been my experience at Royal Prince Alfred Hospital, and means that the group of cases reported on by Dr. Schlink and Dr. Chapman are not fully representative of the cases coming to the hospital, and in that sense are not absolute statistics.

It is, of course, quite impossible for me to present statistics in this disease from Royal Prince Alfred Hospital, as these cases do not come under my care for radium. I have charge of the radium department at Royal North Shore Hospital, and although the numbers are small, the results are very gratifying. As far as I know it is quite impossible for any radiotherapist in Sydney to publish results, as they just do not see the early cases in sufficient numbers.

The clinical research work conducted by Dr. Schlink and Dr. Chapman in exhaustive histological examination of

excised tissues after radium is to be commended; but its value would have been much enhanced if the biological effect had been correlated with the dose of radiation at various points in the pelvis. Their remarks about the measurement of radium dosage in röntgens being liable to an error of *plus* or *minus* 10% are incorrect. Much greater accuracy is now possible, and we have a method by which dosage by various workers can be compared. The actual dose from the same number of milligramme hours in two different cases may vary over an enormously wide range. Therefore milligramme hours has been discarded by modern workers as a unit of dosage.

With respect to ten year figures, the fact is that the modern methods of radiotherapy—and by that is meant an integrated course of treatment by radium and deep X-ray therapy in proper conjunction—have been in extensive use for little longer than ten years, and it is therefore premature to expect ten year results based on sufficient numbers of cases to be statistically valuable.

I am convinced that sufferers from this disease will receive the greatest benefit when cases are centralized into an institute where the appropriate advantages of radio-therapeutic and surgical methods can be applied by experts in these respective fields working in harmonious cooperation.

Yours, etc.,

HAROLD J. HAM.

135, Macquarie Street,
Sydney,
November 18, 1944.

GELATINE IN THE TREATMENT OF SHOCK.

SIR: In the Current Comment entitled "Gelatin in the Treatment of Shock", published in your issue of November 18, reference is made to the work of W. M. Bayliss on gum acacia, and the reader might easily get the idea that gelatin had not been investigated by Bayliss in this connexion. As one who was closely associated with W. M. Bayliss (we were assistant professors of physiology at the same time in University College, London), I should like to point out that the possibilities of gelatin had been fully explored by him, but unfortunately the gelatin on the market was found to contain spores of tetanus bacilli, and Bayliss discovered that heat treatment sufficient to make the gelatin absolutely harmless so reduced its viscosity that it was of little use in combating shock, and in consequence he reluctantly fell back on gum acacia. Some of my old pupils may possibly recall that I frequently demonstrated in the experimental animal the restoration of a good arterial pressure after hemorrhage by gelatin injection, but I always gave the unhappy history of those early attempts to use this procedure in the human being.

Yours, etc.,

W. A. OSBORNE.

The Hall,
Kangaroo Ground,
Victoria.
November 21, 1944.

WHAT THE PEOPLE SHOULD KNOW ABOUT THE TRAGEDY OF TUBERCULOSIS.

SIR: In your issue of November 11 you published a paper by Dr. Sewell entitled "What the People should Know about the Tragedy of Tuberculosis".

One is in complete agreement with Dr. Sewell that the public should be taught something about the disease, but there are several principles that should be rigidly adhered to.

1. Any information given should be scientifically accurate, or should at least be in line with modern scientific investigation.

2. It should be given in such a way that it cannot be misunderstood. People are apt to carry away the most curious notions even after listening to accurate information.

3. Before any restrictions are placed on the tuberculous sufferer, there should be good scientific evidence of the value of such restrictions. This is the most important principle of all.

To be shunned by his fellows is the worst that can befall the patient. If people are given to understand that tuberculosis is as infectious as influenza, that the tubercle bacillus is practically indestructible, and that tuberculosis can be transmitted even by eating and drinking utensils, the tuberculous patient will quickly become an outcast, and will

remain one. If people are led to believe that any room or house that the patient has been in is practically uncleanable, the patient will soon find himself without a roof over his head. This misconception is already a source of considerable hardship to patients.

The evil is twofold. The unfortunate patient is morally branded and set apart, and the result is that cases of tuberculosis will have every incentive to remain undiagnosed. And, as Dr. Sewell himself has remarked, the undiagnosed case is the greatest danger.

If the patient understands that it is generally the cough that infects, he will willingly take such necessary precautions as not coughing into the air in the presence of other people, particularly of children. If he is about to have a fit of coughing, unless he is in bed, it is usually easy to move out of doors till it is over. And if he realizes that sputum also plays a part in the spread of infection, he will readily dispose of his sputum down the sewer, a safe, clean and simple procedure.

Keeping separate eating and drinking utensils is psychologically pernicious, and is a restriction we have no moral right to impose unless we can definitely establish that tuberculous infection is transmitted in this way. After all, even the most inelegant of eaters and drinkers do not slobber much over their utensils. Any bacteriologist can tell how difficult a task it is to find tubercle bacilli in the saliva, and in ordinary washing, any residual saliva is diluted to an extremely high degree in hot soapy water. Further, all the evidence is against the alimentary tract as a source of infection in human pulmonary tuberculosis, especially when the infecting dose would be of the order of but one poor lone emaciated tubercle bacillus that has survived the dish-water.

If infected people are to come forward for supervision and treatment, any restrictions we place on them must be humane and reasonable, otherwise we will defeat our object.

Yours, etc.,

O. E. NICHTERLEIN,
Medical Superintendent, Kalyra
Sanatorium, South Australia.

Undated.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

The annual meeting of the Australasian Medical Publishing Company, Limited, was held at the Printing House, Seamer Street, Glebe, New South Wales, on September 20, 1944, Dr. T. W. Lipscomb, the chairman, in the chair.

Directors' Report.

The report of the directors of the company was as follows:

The directors submit their report for the twelve months ended June 30, 1944, together with the balance sheet as at June 30, 1944, and the profit and loss account for the twelve months ended June 30, 1944.

During the year THE MEDICAL JOURNAL OF AUSTRALIA published many articles from members of the services on war-time medicine and surgery. Other contributions have covered a wide range of subjects. Paper rationing regulations have been eased to a certain extent, and additional pages of reading matter in the journal will be published in the forthcoming year.

The output of the printing and publishing department can be regarded as satisfactory under the circumstances, and despite wartime conditions the work produced was of the usual high standard. It is anticipated that greater supplies of paper will soon be available, but the company will be faced for some time with difficulties in regard to manpower.

The minor alterations to the Articles of Association placed before the members of the company at an extraordinary general meeting of members held on August 27, 1943, were approved by the meeting. They have been embodied in the company's Articles of Association.

Depreciation amounting to £908 was written off during the year, and an amount of £1,191 has been provided for taxation. The company's reserves are used in the business, and we consider the state of the company's affairs is satisfactory.

Provision has been made for the payment of debenture interest for the year ended June 30, 1944.

Dr. T. W. Lipscomb and Dr. J. P. Major retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

August 18, 1944.

T. W. LIPSCOMB,
Chairman.

Election of Directors.

Dr. T. W. Lipscomb and Dr. J. P. Major were reelected to the Board of Directors.

Obituary.

HAROLD OCTAVIUS LETHBRIDGE.

WE are indebted to Dr. Stratford Sheldon for the following appreciation of the late Dr. Harold Octavius Lethbridge.

The sudden and unexpected death of Dr. Harold Octavius Lethbridge was a shock and disappointment to his friends. Knowing for some years that he had an enchondroma on the back of his sternum, he was compelled by its enlargement and the consequent dysphagia to have it removed a fortnight before his death. Apparently well recovered and happy, he suddenly got a pain in the right side of his chest and died, probably from pulmonary embolism.

Harold Lethbridge, known to all his intimates as "Lether", was a remarkable man. His outstanding characteristics were, perhaps, not those of genius, though he had the capacity for taking infinite pains; but rather, he had and practised unselfishly the ordinary human virtues, was devoted to his work, and made this world a more pleasant one for all with whom he came in contact. Some years ago it was said to me by a revered senior practitioner that Lethbridge was the best doctor in Australia. Whether this was so or not, I have never known anyone so proficient and versatile in so many branches of his profession. No doubt anticipating the possible fatal issue, just prior to his operation he wrote of himself: "There must be few who have had a life so full of interest and variety as has been granted to me. Until the last few years an amazing energy drove me from one interest to another, and I have followed them with enthusiasm. Born on the Maranoa, west Queensland, on my father's cattle station, we learnt to

Sit on a bucking brumby
Like a knob on an easy chair
And carve our name in greenhide
On the flank of a flying steer.

We hunted with the black boys, learned bush lore, how to eat grubs and possums and climb trees, also to appreciate the worth of this vanishing race. We joined their corroborees and knew their songs well. . . .

Entering into medicine at the University of Sydney, he "stroked" for Saint Paul's College four times and twice for the university in Melbourne and Adelaide. He became a resident medical officer at Prince Alfred Hospital and later at the Children's Hospital, Glebe Road, and then the superintendent of the latter hospital when it moved to its present site.

Desiring to practise in the bush, he went to Narrandera, first as assistant, and later to become the doyen of the medical profession in the Riverina. A "horse and buggy doctor" in a very large district, he became an experienced general practitioner. He soon showed that he was a skillful and shrewd surgeon, with natural and acquired dexterity. Benefiting by his early experience in hospital, he did his own pathology and bacteriology. He maintained that a hospital without a laboratory was only a boarding-house for the sick, and so set about establishing these departments in Narrandera; he conducted them all himself and trained assistants. The same applied to radiology. In the last few years he had more or less retired from general practice, devoting his time to these branches of medicine. It was an education to observe him each morning working with his assistants at Narrandera Hospital, and doing not only the hospital's own work, but also that from surrounding institutions. He was much valued as a consultant, as he was helpful without being patronizing. His enthusiasm was infectious; he always promoted good feeling among his colleagues, and his eponym "Lethbridge House" indicates that he was not without honour in his own town.

He served in the first world war. After some time in the Middle East, he was appointed to base hospitals in

Italy and Sicily, and later to the orthopaedic hospital in England. He endeared himself to soldiers as much by his entertaining them with his various accomplishments as by his assiduous professional attention. He never allowed this interest and connexion to lessen, and for the rest of his life returned soldiers were his pets. His great interest in life apart from his profession, or rather combined with it, was his care for study of the Australian aborigines. As he said of himself, he knew their lore and life; he had the genealogical trees of several families tabulated for three generations. He wrote several articles on this subject; his conclusion was that, as their race became mixed with the white, the white, being dominant, gradually eliminated the black. For thirty odd years he was the father as well as the professional guide to the local blacks' reserve. He was much interested in Australiana generally; his numerous collection of weapons, fossils and anything pertaining to Australian history was presented to the Department of Public Instruction, and is now housed in a special annexe to the local school. His enthusiasm has induced numerous friends to present historical objects and documents, which are added to the museum. He himself was a true Australian; his progenitor in Australia settled here shortly after the arrival of the first fleet under the command of Governor Phillip. The striking feature of his career is the wonderful influence a modest and unselfish but enthusiastic life had on the community in which it was spent—it added much to its well-being and culture. To be looked to as the Nestor of his community and profession was his reward, and must have brought him great satisfaction. Farewell.

Naval, Military and Air Force.

CASUALTIES.

ACCORDING to the casualty list received on December 2, 1944, Major J. S. Chalmers, A.A.M.C., Hobart, who was previously reported "prisoner of war", is now reported "missing, believed deceased".

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Bonnette, Stanley Albert, M.B., B.S., 1937 (Univ. Sydney), NX106995, Major S. A. Bonnette, 2 Australian Field Ambulance, Australia.
Segal, Harry, M.B., B.S., 1942 (Univ. Sydney), 9, Niblick Street, North Bondi.
Hammond, Brian Thomas, M.B., B.S., 1941 (Univ. Sydney), 127, Belgrave Street, Kempsey.
Robey, Lancelot Turbet, M.B., B.S., 1942 (Univ. Sydney), NX201399, Captain L. T. Robey, 1 Papuan Infantry Battalion, New Guinea.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Ewart, Charles Colin, M.B., B.S., 1942 (Univ. Sydney), NX201400, Captain C. C. Ewart, 2/15 Field Ambulance, A.I.F., Australia.
Frith, Alexander Richard, M.B., B.S., 1942 (Univ. Sydney), Captain A. R. Frith, P.O. Box 251, Lismore.
Gibson, Lloyd Cameron, M.B., B.S., 1943 (Univ. Sydney), District Hospital, Wollongong.
Hall-Johnston, John, M.B., B.S., 1937 (Univ. Sydney), Squadron Leader J. Hall-Johnston, Group 675, R.A.A.F., Pacific.
Howse, Neville Beresford, M.B., B.S., 1942 (Univ. Sydney), Flight Lieutenant N. B. Howse, 68, Sampson Street, Orange.
Irvine, Allan Francis, M.B., 1943 (Univ. Sydney), 194, Paine Street, Maroubra.
Kennett, Arthur Lloyd, M.B., B.S., 1939 (Univ. Sydney), Squadron Leader A. L. Kennett, "Hazelwood Park", Glenfield.
Laing, John, M.B., B.S., 1942 (Univ. Sydney), Captain J. Laing, 233, O'Sullivan Road, Bellevue Hill.
Laver, Eric Geoffrey, M.B., B.S., 1943 (Univ. Sydney), 37, Bancroft Avenue, Roseville.

Medical Appointments.

Dr. Lachlan Martin McKillop, Dr. Henry Joseph Windsor (nominated by the Minister to represent the Government), Dr. Felix Wilfred Arden, Dr. David Gifford Croll and Dr. John George Wagner (nominated by the Queensland Branch of the British Medical Association, recognized by the Minister as representative of medical practitioners), in pursuance of the provisions of *The Medical Acts, 1939 to 1940*, of Queensland, have been appointed members of the Medical Board of Queensland.

Dr. Alan Allison Barr has been appointed Government Medical Officer at Miles, Queensland.

Dr. John Edmund Thomas has been appointed a Public Vaccinator, Victoria.

Books Received.

"Salts and Their Reactions: A Class-book of Practical Chemistry", by Leonard Dobbin, Ph.D., and John E. Mackenzie, O.B.E., D.Sc.; Seventh Edition; 1944. 8½" x 5½", pp. 253. Price: 8s. 6d. net.

"The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; Sixty-Second Year, 1944. 8½" x 5½", pp. 413, with illustrations. Price: 25s. net.

"Minor Surgery", by R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.I.C.S.; Second Edition; 1944. London: H. K. Lewis and Company, Limited. 7" x 4½", pp. 398, with many illustrations, some in colour. Price: 15s. net.

"Lumbo-Sacral Strain: A Handbook on Its Relief and Cure by Manipulative Therapy", by H. Vincent Langley, with a foreword by N. Howarth Hignett; 1944. London: William Heinemann (Medical Books) Limited for Research Books Limited. 7½" x 4½", pp. 24, with illustrations. Price: 6s. net.

"Casualty Work for Advanced First-Aid Students", by A. W. Macquarrie, M.B., Ch.B. (Edinburgh); 1944. Edinburgh: E. and S. Livingstone Limited. 4½" x 3½", pp. 250, with illustrations. Price: 4s. 6d. net.

"Notable Names in Medicine and Surgery", by Hamilton Bailey, F.R.C.S. (England), and W. J. Bishop, F.L.A.; 1944. London: H. K. Lewis and Company, Limited. 7½" x 5", pp. 209, with many illustrations. Price: 15s. net.

"Modern Treatment in General Practice Year Book, 1944: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Cecil P. G. Wakeley, C.B., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. (Hon.); 1944. London: The Medical Press and Circular. 8½" x 5½", pp. 308, with illustrations. Price: 16s.

"Principles and Practice of Aviation Medicine", by Harry G. Armstrong, M.D., F.A.C.P.; Second Edition; 1943. London: Baillière, Tindall and Cox. 9" x 6", pp. 524, with illustrations. Price: 36s.

"A Synopsis of Hygiene (Jameson and Parkinson)", by G. S. Parkinson, D.S.O., M.R.C.S., L.R.C.P., D.P.H., Lieutenant-Colonel, R.A.M.C. (Ret.), with a section on personal hygiene, by G. P. Crowden, O.B.E., D.Sc., M.R.C.S., M.R.C.P., T.D.; Eighth Edition; 1944. London: J. and A. Churchill Limited. 8½" x 5½", pp. 734. Price: 25s.

"Essentials of Syphilology", by Rudolph H. Kampmeier, A.B., M.D.; 1943. Philadelphia: J. B. Lippincott Company. London: Macmillan and Company, Limited. 8" x 5", pp. 534, with 87 illustrations, one of which is in colour. Price: 25s. net.

"Artificial Pneumothorax in Pulmonary Tuberculosis, Including Its Relationship to the Broader Aspects of Collapse Therapy", by T. N. Rafferty, M.D., with an introduction by Henry Stuart Willis, M.A., M.D.; 1944. New York: Grune and Stratton. 8½" x 5½", pp. 208, with 26 illustrations. Price: \$4.00.

"The Electrocardiogram, Its Interpretation and Clinical Application", by Louis H. Sigler, M.D., F.A.C.P.; 1944. New York: Grune and Stratton. 9" x 6", pp. 415, with 203 illustrations. Price: \$7.50.

"Industrial Medicine", edited by Sir Humphry Rolleston, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and Alan A. Moncrieff, M.D., F.R.C.P., with an introduction by Air Vice-Marshal Sir David Munro, K.C.B., C.I.E., M.B., Ch.B., F.R.C.S. Ed., LL.D., 1944. London: Eyre and Spottiswoode (Publishers) Limited. 8½" x 5½", pp. 202, with illustrations. Price: 16s. net.

"Why Singapore Fell", by Lieutenant-General H. Gordon Bennett; 1944. Sydney: Angus and Robertson Limited. 8½" x 5½", pp. 273, with illustrations. Price: 12s.

"L'inhibition et la facilitation dans le système nerveux central et périphérique", par Miguel Otorio de Almeida; 1944. Rio de Janeiro: Atlantica Editora. 9½" x 6½", pp. 136.

"Études de psychologie médicale: I. Perception et langage", par André Ombredane; 1944. Rio de Janeiro: Atlantica Editora. 9½" x 6½", pp. 185.

"Études de psychologie médicale: II. Geste et action", par André Ombredane; 1944. Rio de Janeiro: Atlantica Editora. 9½" x 6½", pp. 129.

"Introducing Australia", by C. Hartley Grattan; 1944. New York: The John Day Company. Sydney: Angus and Robertson. 8½" x 5½", pp. 311, with illustrations. Price: 12s. 6d.

"One Hundred Poems: 1919-1939", by Kenneth Slessor; 1944. Sydney: Angus and Robertson Limited. 7½" x 5", pp. 131. Price: 5s.

Diary for the Month.

- DEC. 11.—Victorian Branch, B.M.A.: Executive Meeting.
- DEC. 12.—Tasmanian Branch, B.M.A.: Branch Meeting.
- DEC. 12.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- DEC. 13.—Victorian Branch, B.M.A.: Council Meeting.
- DEC. 14.—New South Wales Branch, B.M.A.: Branch Meeting.
- DEC. 15.—Queensland Branch, B.M.A.: Council Meeting.
- DEC. 15.—Queensland Branch, B.M.A.: Annual Meeting.
- DEC. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
- DEC. 21.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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